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DELAWARE STATE MEDICAL JOURNAL

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VOLUME 29 JUNE, 1957 NUMBER 6

DIAGNOSIS AND TREATMENT OF DISEASES OF THE EYE FOR THE G.P.

Complete Contents on Page iv

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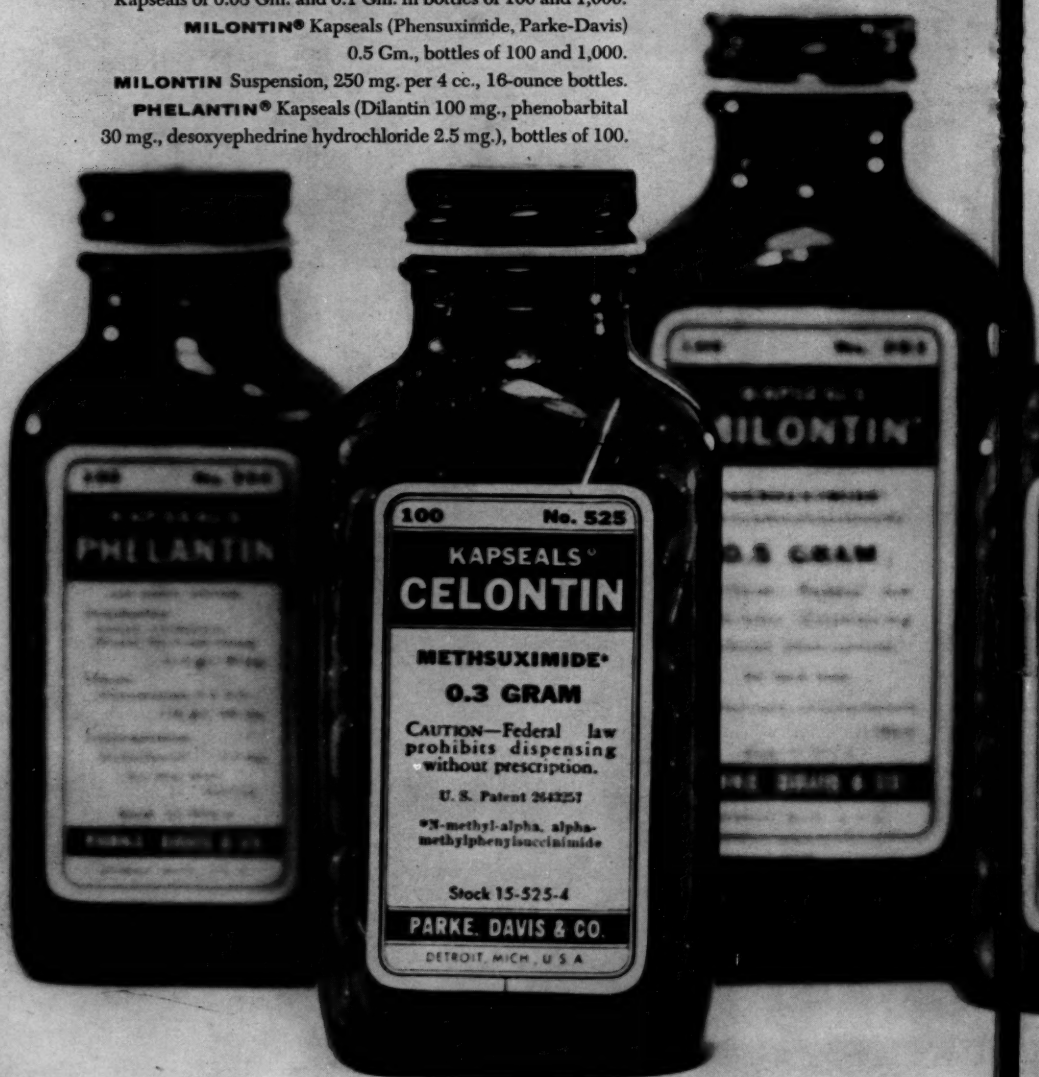
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1. Zimmerman, F. T., and Burgemeister, B.: *Arch. Neurol. & Psychiat.* 72:720, 1954.
2. Zimmerman, F. T., and Burgemeister, B.: *J.A.M.A.* 157:1194, 1955.
3. Zimmerman, F. T.: *Arch. Neurol. & Psychiat.* 76:65, 1956.

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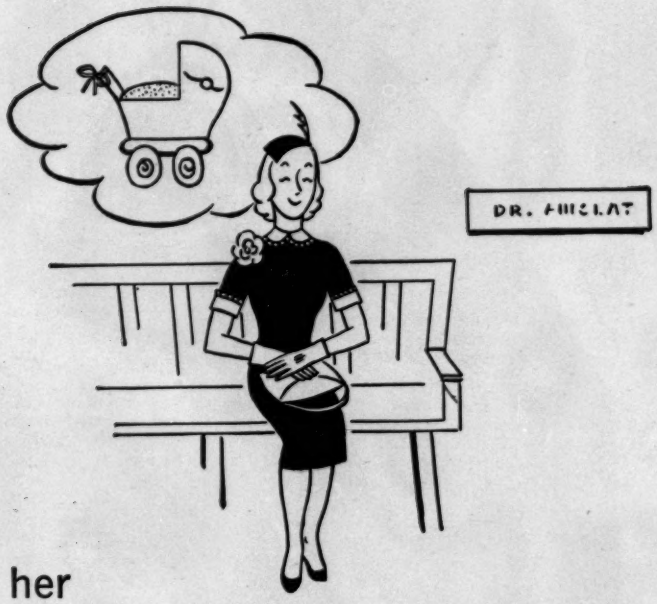
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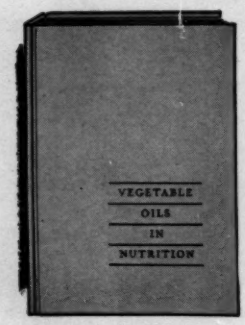
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Keyes, B. L.: Pennsylvania M. J. 60:177, Feb. 1957.

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Sokoloff, O. J.: A.M.A. Arch. Dermat. & Syph. 74:393, Oct. 1956.

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Kessler, L. N. and Barnard, R. D.: M. Times 84:431, April 1956.

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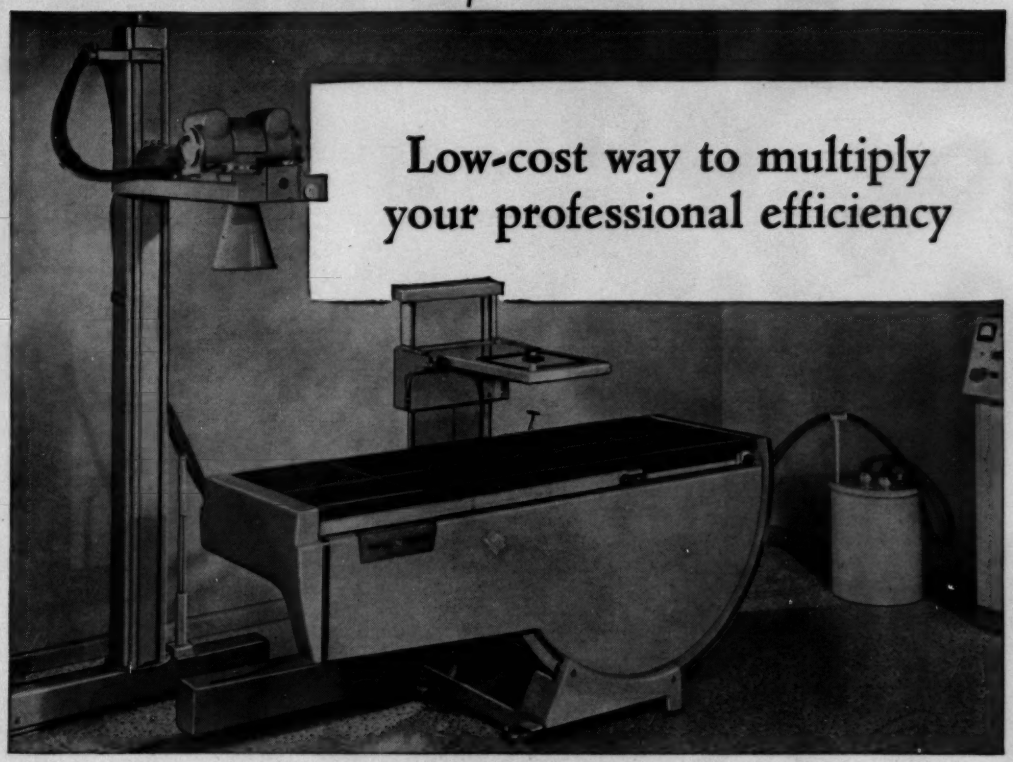
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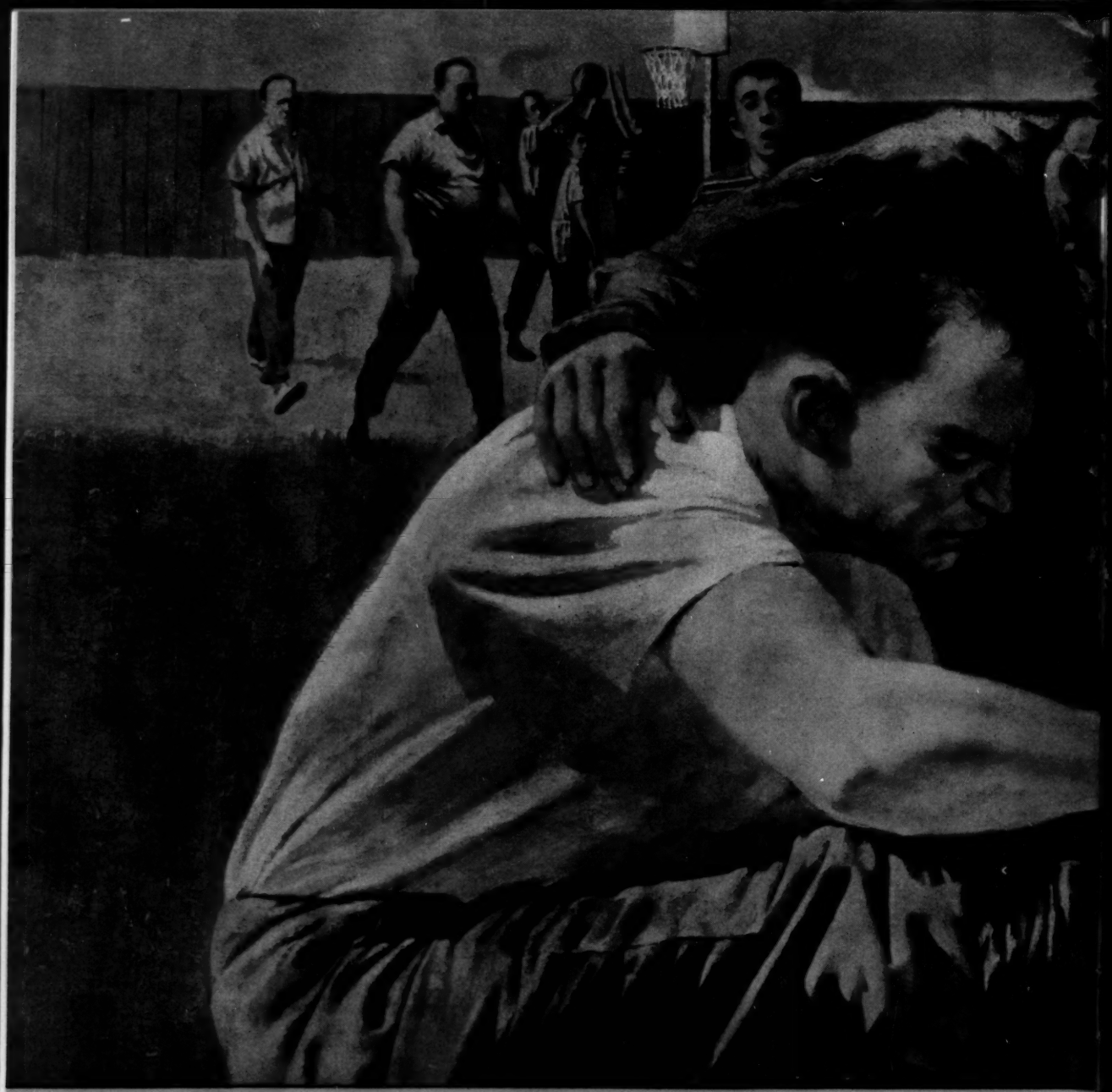
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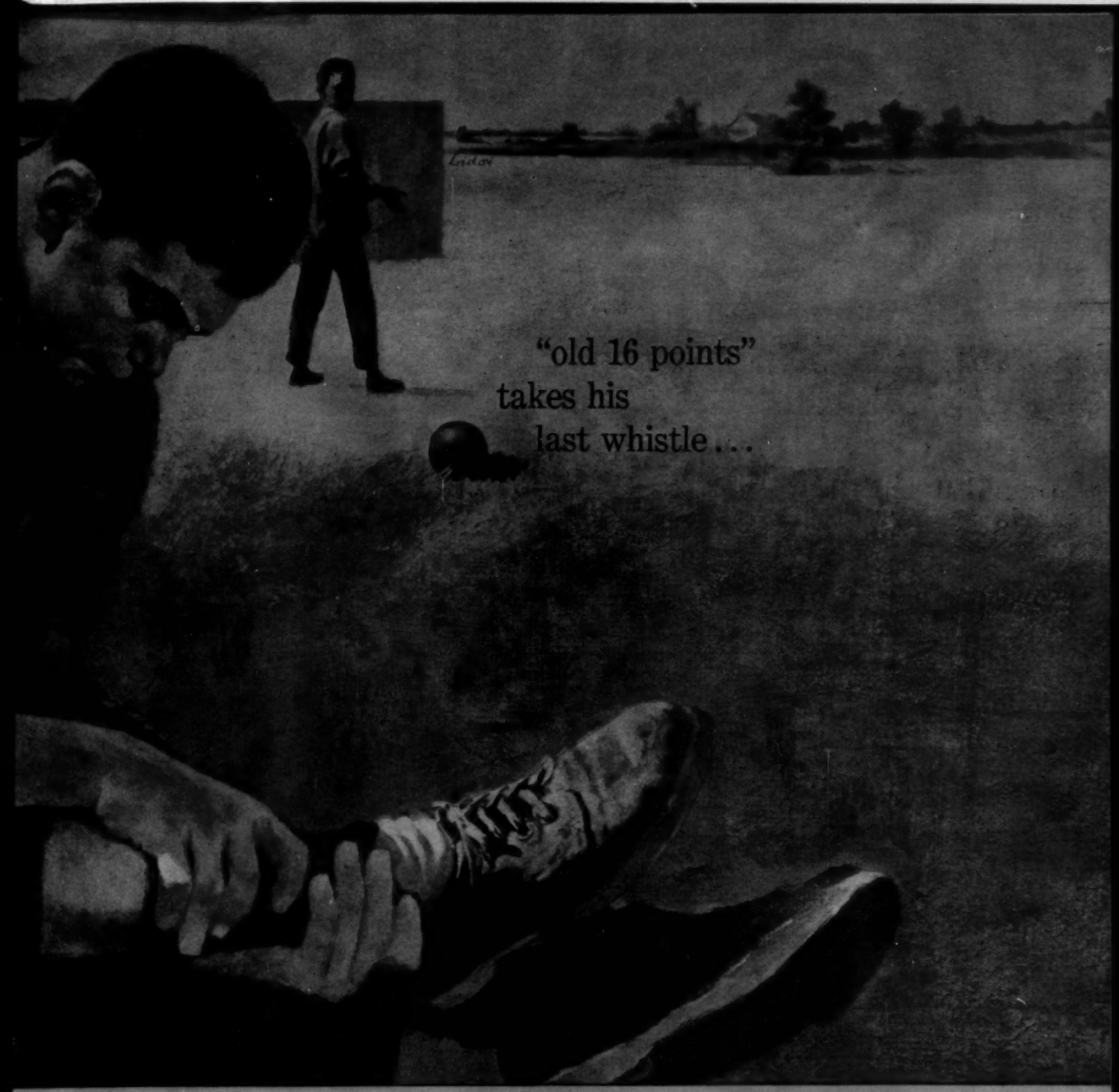
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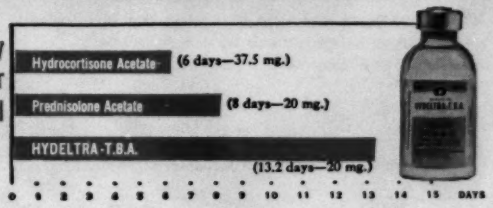
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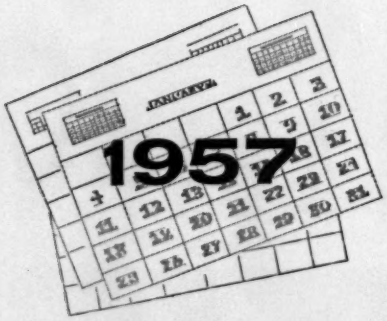
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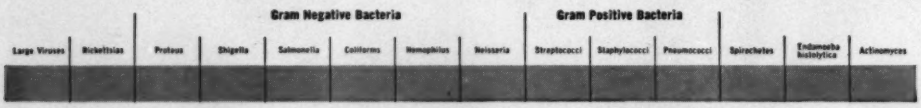
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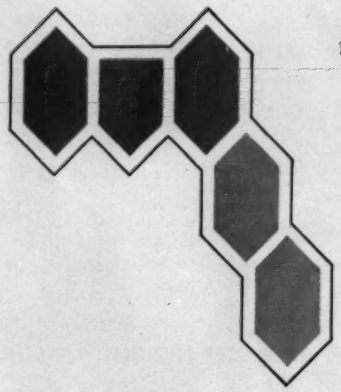
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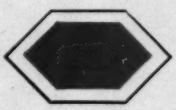
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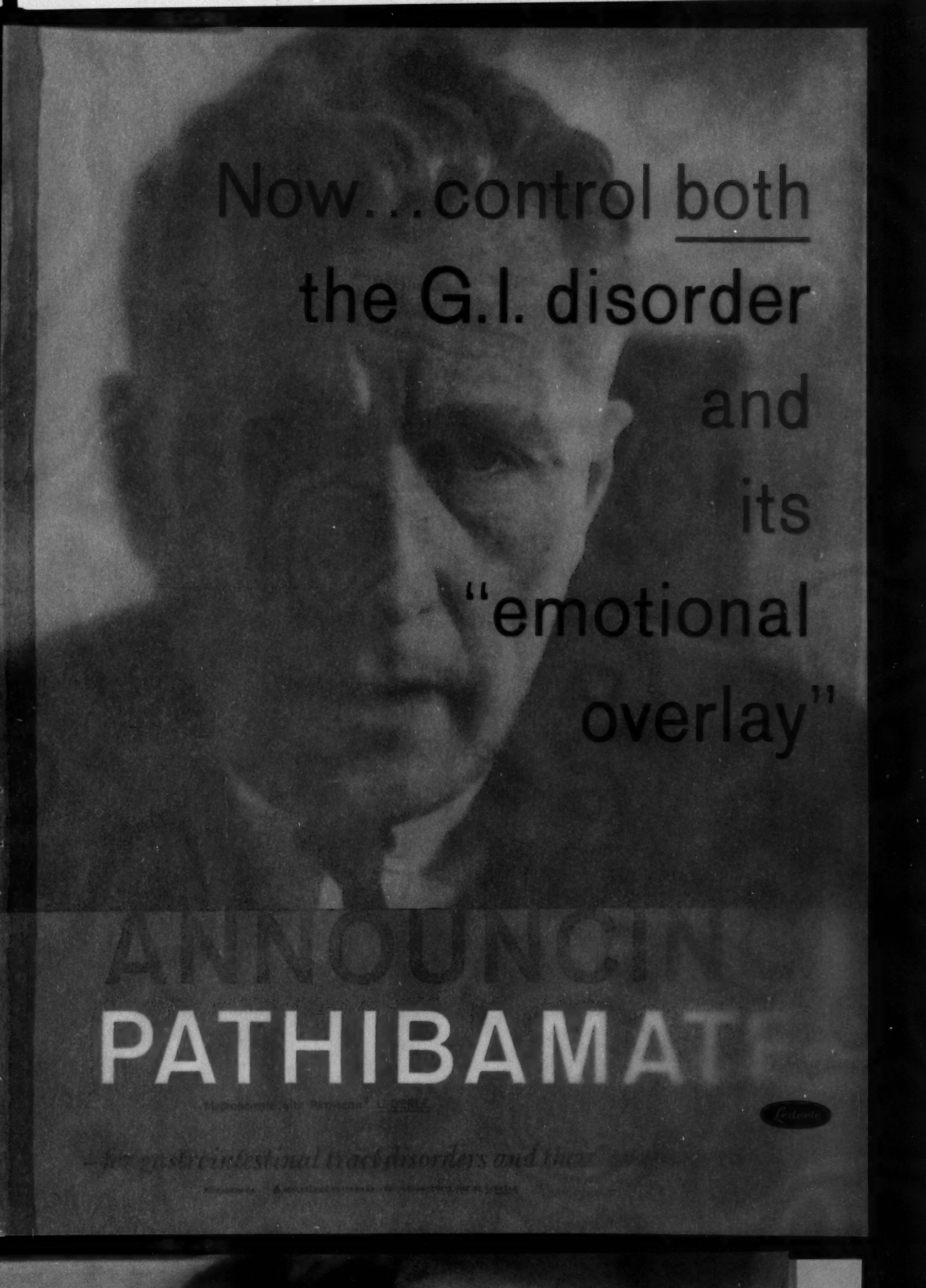
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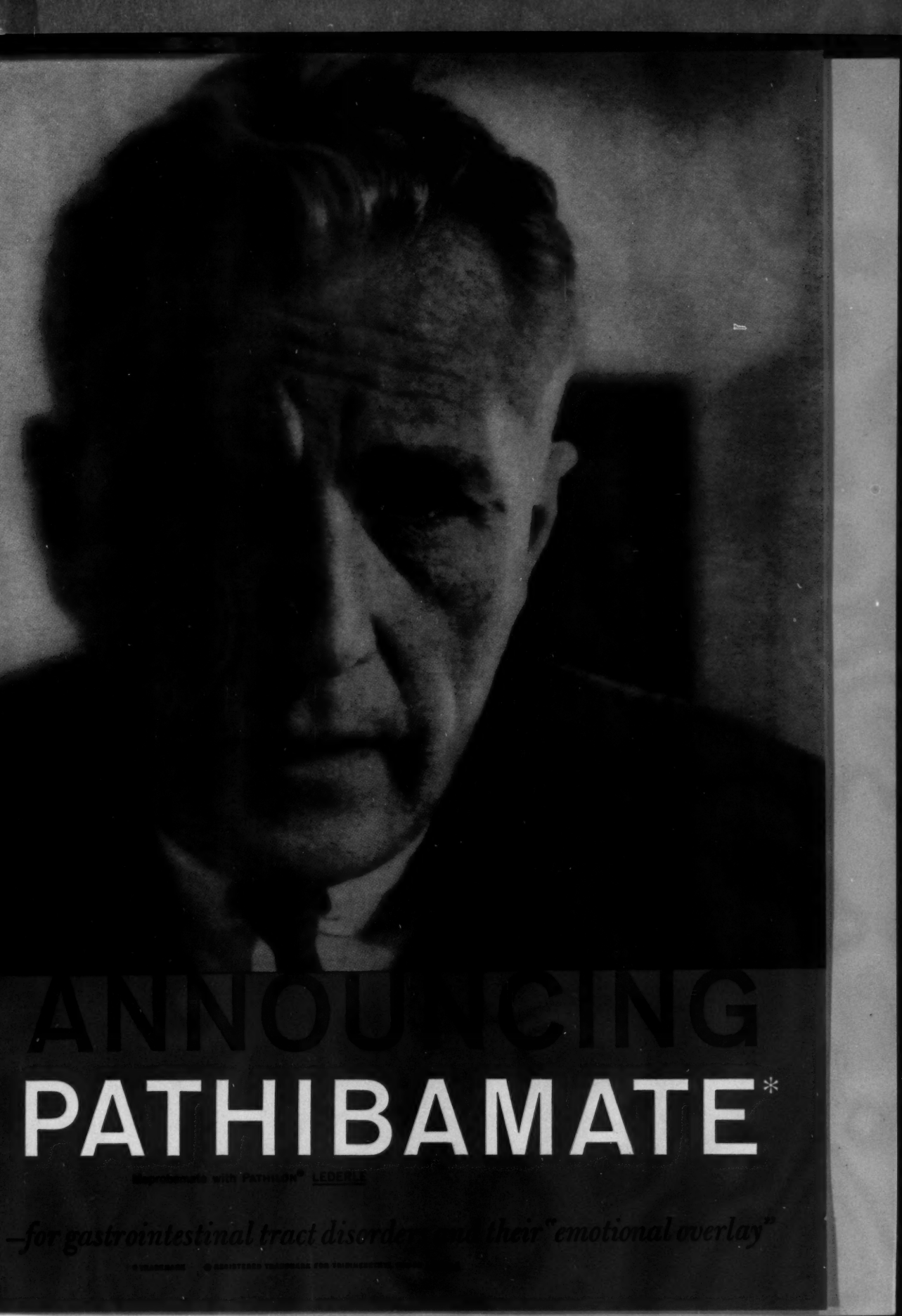
- Patients experiencing ... reserpine often were ... when changed to Harmonyl.

Ferguson concluded: "... impressions were the ... and relatively rapid ... Harmonyl."

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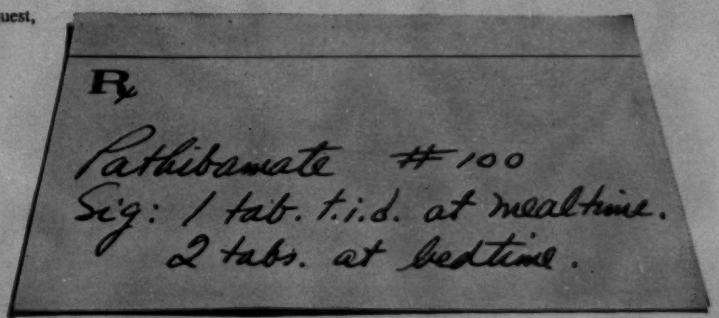
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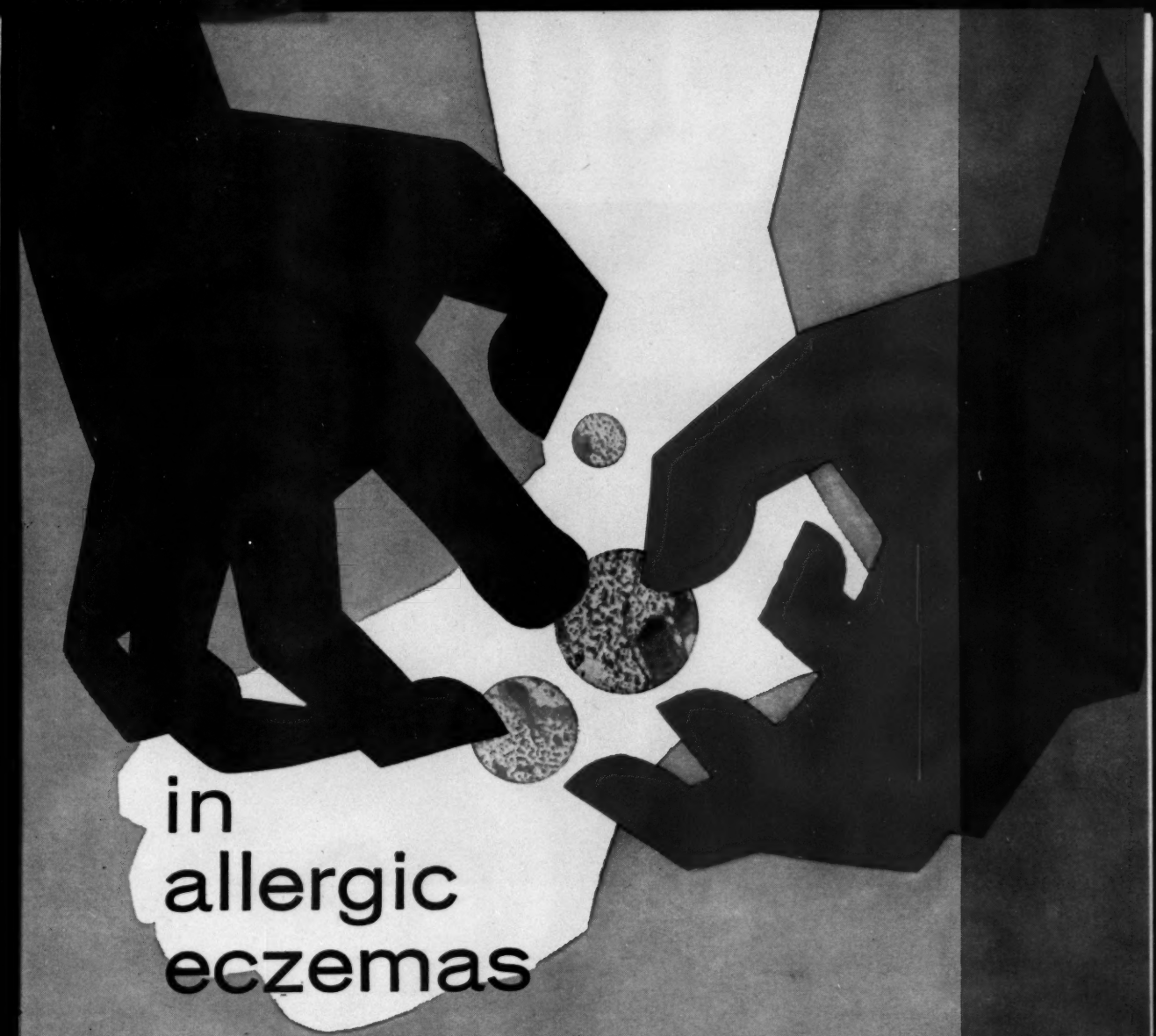
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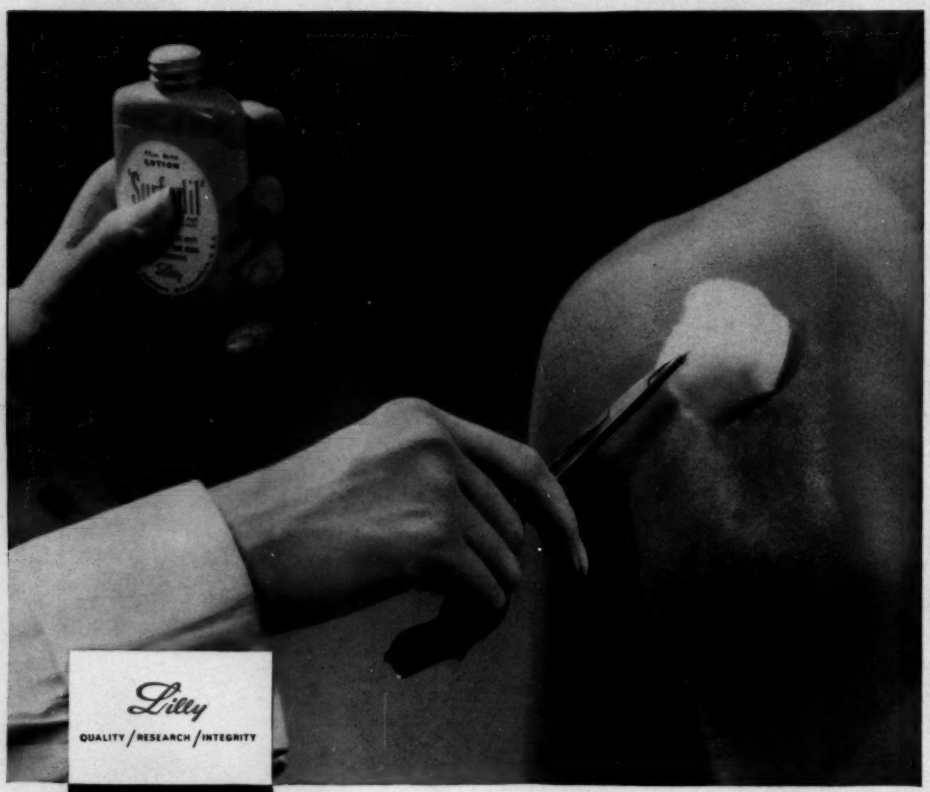
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DIAGNOSIS AND MANAGEMENT OF OCULAR PROBLEMS

by

WILLIAM H. KRATKA, M.D.*

This article is intended to discuss most of the ocular problems that confront the general practitioner. Discussion has been kept to a minimum and the references have been selective rather than complete.

OUTLINE

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I. ROLE OF THE FAMILY PHYSICIAN

The family physician plays a vital role in the education of his patients in proper eye care and the prevention of blindness.

To assist the medical profession and the public with pertinent information about the care and treatment of the eye, this year has seen the establishment of the National Medical Foundation for Eye Care, Inc., organized by the nations' ophthalmologists.

At the March meeting of the American Academy of General Practice,[1] the National Society for the Prevention of Blindness in its exhibit demonstrated the following facts

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in cognizance of the importance of eye care:

1. Over one third of a million Americans are totally blind.
2. Another million are blind in one eye.
3. Approximately 750,000 now enjoying sight will become blind, half of them needlessly.

Against these negative statements are reported the following gains:

1. Ophthalmia neonatorum has been cut by 98%.
2. Children's eye injuries have decreased by 37%.
3. Blindness from infectious diseases was cut 48% in children and 23% in adults.
4. A total of 8,000 American babies have been blinded by retrolental fibroplasia, now a preventable disease.

Today we face the following problems:

1. A longer life span is causing an increased prevalence of cataracts, responsible for one fifth of adult blindness; glaucoma is responsible for one eighth.
2. Over a million Americans above the age 40 have early signs of glaucoma.
3. Diabetes has become an important predisposing factor to blindness.

II. VISION

Vision is a highly complex sense.[2] To produce a clear image in consciousness there takes place an integration of three systems, i.e. (1) the visual pathway; (2) eye movement and binocular balance; and (3) the higher centers. Impairment of vision may result from interruption of any of these systems. The retina acts as the receiver for light impulses entering the cornea. These become nerve impulses and continue through the optic nerve, optic chasm, optic tracts, optic radiations, and finally arrive in the visual cortex and higher centers. Thus the statement "we see with our brains, not with our eyes alone".

A—*Visual examination of children:* This requires some knowledge of visual development as well as skill and patience. Up to the age of five the following are considered normal in the stages of development of the pre-school child:

1. Full term newborn: has pupillary movements, darkness and lightness are comprehended.
2. First month: Baby will watch a bright light and within two foot range will follow an object through a 90° arc.
3. The 3rd and 4th months: Baby has fairly good development of convergence and the following of movements.
4. 9 months: Depth perception starts, the infant can play with tiny objects.

Range of Visual Acuity:

4 months—20/400
1 year—20/200
2 years—20/40
3 years—20/30 (picture chart)
4-5 years—20/30 (E chart)

Testing of Vision during infancy: Here we depend on the pupillary light reaction; and we note whether the child blinks at bright light and can follow bright moving objects. Acuity is first tested at 2-3 years of age: a 1½ in. marble may be used and if recognized at 20 feet vision is about 20/200; seeing ¾ in. wide marble at 20 feet means about 20/80 vision; at 3 to 4 years use is made of the animal kindergarten charts and at 4 to 6 years the Snellen E chart.

School Vision Screening: Vision screening of school children has been conducted by many screening methods.[3] The simpler and more efficient the device, the more accurate have been the results. The Massachusetts Vision Test (MVT) has failed to prove more trustworthy than the fundamental Snellen test. The subsidiary tests of the M.V.T. i.e., the plus 1.50 to 2.00 diopter sphere and the Maddox rod-prism measurement produce an additional small group of cases which merit care but do so with a high rate of over-referrals. By the Snellen test, failure to read 20/30 with either eye is used as the standard of referral. This simple and efficient method discloses from two-thirds to three-fourths of all cases professionally meriting care.

General observation by the examiners for gross signs of pathology (ptosis, strabismus, etc.); signs of stress on visual performance (squinting, peering, head tilt, etc.); and symptoms or complaints referrable to vision (pain, blurring, double vision) should be noted to supplement the Snellen test.

B. *Industrial Vision:* Here again we find best results being obtained by testing at 20 feet with the Snellen chart and at 13 inches with the Jaeger test type. Complicated methods such as telebinoculars and ortho-rators, while assisting in screening for vision, depth perception, and ocular motility fail to attain the accuracy of the simpler tests. These methods also require accurate performance and well trained technicians. Proper lighting for plant work should be carefully evaluated in regard to the type of work required and should not be over illuminated, particularly by fluorescent lights alone. Glare and light reflections will frequently cut down industrial and office efficiency as well as initiate many symptoms of ocular fatigue.

III. STRABISMUS

Six extra-ocular muscles move each eye, (seven counting the eyelid). In each eye these muscles are controlled directly by the third, fourth and sixth cranial motor nerves and by complicated associated nerve tracts in the brain areas. Visual function can be upset by a disturbance of a single muscle or of the motor nerve supplying it. Any defect in binocular balance and vision results in blurring, headaches or, if more pronounced, double vision.[2]

When the tendency of an eye to deviate (phoria) becomes intolerable, binocularity is lost, and the result is outright strabismus or squint. Unfortunately, most non-paralytic squints begin between ages one and four, before school age. Our aim in treatment should be to diagnose and treat these problems as soon as possible. Besides the cosmetic defect plus the loss of binocular and stereoscopic vision, there are more severe disabilities from strabismus including amblyopia ex-anopsia due to visual suppression in the squinting eye plus emotional disturbances and behavior problems. In no instance should the parents be allowed to think the child will outgrow the strabismus.

Evaluation of strabismus requires: (1) Determination of visual acuity particularly noting inequality of the two eyes as a possibility of suppression amblyopia. (2) A measurement of the degree of squint made either by the Hirschberg method in noting the deviation of the light reflexes from the center of squinting eye or by more refined techniques with prisms or an orthoptic examination. (3) The child's eyes are refracted under cycloplegia and glasses are prescribed where indicated.

Treatment of strabismus consists of refraction, surgery (where indicated), and in many cases, orthoptic treatment. Orthoptics alone does not straighten eyes. Its purpose is diagnostic as well as the re-education of the eyes in the lost art of binocularity. Defective vision in the squinting eye requires patching of the good eye until vision in both eyes is approximately equal; occasionally drops and partial occlusion may be used for this purpose. This is most effective in late infancy and early childhood. Let me once again emphasize that early treatment of strabismus gives the best results. Where possible major correction should be made before school age both to prevent emotional upsets as well as to best assist the development of the fusion mechansm.

IV. GLAUCOMA

Blindness has been known throughout the ages. Hippocrates in his "Aphorisms" mentioned a disorder known as "glaucosis" marked by a greenish-or-bluish-gray pupil, as a cause of blindness. Galen noted glaucoma to be the most common cause of blindness. Glaucoma may be defined as an abnormal elevation of intraocular pressure which is incompatible with normal function of the eye. Vision is lost permanently because of the pressure on the nerve fibers entering the optic disk. Normally, intra-ocular tension ranges between 18 to 25 mm. of mercury with Schiötz tonometer. Mass tonometric screening reveals that 2% of our population over 40 years

of age have undetected glaucoma.[4] Any patient within this age group complaining of blurred vision, ocular pains, or halos about lights at night should be suspected of having this debilitating disease. Routine tonometry should be a part of all medical examinations over the age of 40. Tonometry by simplified methods must be taught and emphasized to medical students and internes. Only with the aid of early tonometry and rapid visual field testing can this surreptitious disease process be prevented from becoming a serious menace to our increasing and aging population.

Types of Glaucoma: Glaucoma is divided into (a) primary types, i.e. narrow angle and open angle, (b) secondary type, i.e. principally inflammatory, traumatic and cataractous, and (c) congenital form (Buphthalmis).

In the treatment of glaucoma the ophthalmologist must differentiate between the acute congestive, narrow-angled form of glaucoma characterized by bouts of severe pain plus a rapid rise in pressure; in contrast to the simple, wide or open angle type in which ocular hypertension develops insidiously over the years. It is principally in the simple variety with consistently elevated tension that we find progressive cupping of the optic disk and loss of visual fields.[4]

Therapy of glaucoma includes frequent visual evaluation, tonometric and tonographic tension studies, visual field examinations, and gonioscopic examinations of the anterior chamber angle.[5] Where possible the control of tension as well as the maintenance of the visual fields is attempted by means of miotics such as Pilocarpine, Doryl, and D.F.P. (Floropryl) as well as Diamox. In contrast to the chronic form, acute narrow angle glaucoma, if operated upon early, usually can be cured by an iridectomy, which will repair the block of the chamber angle. In the chronic forms, surgery is indicated only when medical therapy is unsuccessful in maintaining normal ocular tension, vision, and visual field.

V. OCULAR INFECTIONS

A—*Blepharitis:* (or infections of the lids) Blepharitis represents one of the commonest forms of ocular pathology. Ophthalmologically we separate these infections into two types:[6]

1. *Eczematous Blepharitis*, contact dermatitis (allergic): Treatment and diagnosis in these cases is aimed at searching for and eliminating the possible allergens. These may consist of topically applied drugs, cosmetics, pollens, dust and chemicals. Blepharitis varies in severity from the acute edematous and ulcerative form to the chronic dry parchment-like phase. Besides elimination of the offending agent, local cortisone, antihistamines, plus systemic ACTH and cortisone is the present treatment of choice. In some of the more stubborn cases a series of intracutaneous tests are done as part of the investigation.
2. *Chronic Blepharitis:* Here we find variation in type and appearance; from the greasy, dandruff like scaly seborrheic form; to the more irritative, ulcerative and pustular variety. Etiologically we find the Pityrosporon ovale organism causing the seborrheic type; whereas in the ulcerative lesions we mostly find the hemolytic staphylococci.

Therapy consists of local antibiotic applications to the cilia and lid margin.[7] Thygeson recommends selenium sulfide ointment for the seborrheic form, although this may be found very irritating if contact occurs with the eye. In resistant cases, especially when accompanied by scalp infections, treatment of the seborrhea will require salicylic and mercurial ointments.

In ulcerative blepharitis, besides local antibiotic and sulfonamide therapy, desensitization and immunization with staphylococcus toxoid and vaccine should be considered. The use of hot compresses of hot water or boric acid solution should not be neglected to reduce the acute irritation of the lid margins.

B—*Conjunctivitis:* Viruses: By far the most significant conjunctival literature during the past year is that concerning the so-called APC or adenoidal-pharyngeal-conjunctival group of viruses (adenoviruses). Serologically these have been subdivided by specific neutralization tests into serologic types. Conjunctival irritation may vary from a follicular form to the fulminating epidemic kerato-conjunctivitis type. Many of these cases have proved secondary to swimming pool exposure and are now termed pharyngoconjunctival fever. Whereas antibiotics and sulfonamides have no therapeutic value here, steroids, locally, lead to the relief of symptoms.

Baterial conjunctivitis: In resistant cases it is important to discover the etiologic factor and determine the sensitivity of the organism to antibiotic therapy and sulfonamides. As in all fields of medicine, new agents are constantly being tried. Many staphylococci are proving very resistant to tetracycline therapy.

C—*Herpetic Keratitis* (Dendritic ulceration): Caution in the treatment of these virus ulcerations of the cornea should be exercised. Dendritic ulcerations are nonresponsive to cortisone therapy and may end in perforation of the globe. Cortisone suppresses the inflammatory reaction causing a false sense of security. Tissue culture may establish a positive diagnosis as early as 6 to 8 hours after innoculation. These cases are best treated by an ophthalmologist and early cauterization is the best form of therapy.

VI. Eye Injuries and Foreign Bodies

A—*Eyelid lacerations:* To obtain good results, eyelid injuries must be repaired within 24 to 36 hours. Horizontal lacerations of the skin not involving the lid margin can usually be sutured directly by the general practitioner. Suturing is best done with atraumatic needles and black silk (000000). Vertical lacerations require accurate repair (to avoid notch formation at the lid margin) of the tarsus and orbicularis, and are best done by a specialist.[8]

B—*Mechanical Injuries:* Contusions and concussions of the globe are a deceptive group of injuries. Trauma may be so slight as to require no treatment, or may result in perforation of the globe, retinal separation, intraocular hemorrhage, glaucoma, cataract, and sympathetic ophthalmia. Whenever possible, it is always wise to check the patient's vision and examine the interior of the globe with the ophthalmoscope at the time of the accident. These cases should always be carefully examined, usually by x-ray, for possible intraocular foreign body.

C—*Lacerations and abrasions of cornea and conjunctiva:* Small lacerations (up to 2-3 mm.) of the conjunctiva frequently heal without repair. Corneal lacerations require suturing. Treatment of lacerations should include: (1) a careful search for a possible foreign body; (2) installation of an antibiotic; and (3) a pressure patch.

D—*Foreign Bodies:* Most foreign bodies lodging in the eye are contaminated with bacteria and antibiotics should be given as part of the treatment. Detection of a foreign body of the cornea requires good illumination, a binocular loupe, and frequently fluorescein staining. Superficial foreign bodies of the cornea may be readily removed with a sterile eye spud. Deeply imbedded particles and rust rings are best removed by a trained specialist with the help of a slit lamp. Relief from subsequent pain following removal of the foreign body is best achieved by means of ½% Pontocaine or Butyn eye ointment, plus a tightly applied sterile eye patch, to prevent lid irritation.

VII. Chemical Burns of the Eye

First aid for any chemical injury to the eye should consist of immediate and copious flushing with plain water or normal saline. Always flush under the lids to be sure of removal of all particles. Local anesthetic, as ½% Pontocaine, should be instilled to relieve the severe blepharospasm. Fluorescein should be used to stain the cornea and conjunctiva to determine areas damaged. To relieve the marked photophobia and frequent iritis from chemicals, dark glasses and either homatropine

(for mild exposures) or atropine (for severe cases) can be used daily until the marked irritation subsides. Cortisone locally is very helpful in relieving the inflammatory reaction but must be used cautiously at onset so as not to retard the regrowth of the corneal epithelium. Most severe burns of the cornea are frequently complicated by iritis and should be followed carefully with slit-lamp observation. Deeply penetrating chemical burns must be followed closely until all activity ceases. At this time evaluation of vision, corneal scarring, and invasion of vessels should be made. The specialist can then determine the value of beta-radiation to reduce corneal vascularization and the possibility and need of restoring vision by means of superficial keratectomy or a penetrating corneal transplant.

VIII. Diseases of the Retina

Ophthalmoscopic examination is just as important in the diagnosis and treatment of systemic diseases as it is for ocular diseases. If adequate examination cannot be made through a normal size pupil and glaucoma is not suspected, the pupil may safely be dilated by Paradrine 1%, Neo-synephrine 2½%-10% or Cyclogel ½% or 1%.

A—*Retrolental Fibroplasia:* In 1952, oxygen administration to premature infants was implicated as an etiologic agent in the development of retrolental fibroplasia (RLF). The following conclusions were reached by 1956 regarding the major factors affecting this dibilitating disease[9]:

1. The incidence of active stages of RLF in infants receiving routine oxygen was twice that found in those with curtailed oxygen.
2. Both active and cicatricial forms of RLF was less in infants of single birth than those of multiple birth.
3. Increased duration of oxygen exposure rapidly increased the incidence of both active and cicatricial changes.
4. All infants showed an inverse ratio relationship of cicatricial RLF to their birth weight. (Smaller birth weight—greater changes).
5. There is no concentration of oxygen in excess of that which is in air that is not associated with the risk of developing RLF.

Clinical Stages of RLF

1. Vascular stage: Characterized by dilation and tortuosity of the retinal vessels, with areas of neovascularization peripherally.
2. Hemorrhages and transudates stage: These appear around the abnormal vessels.
3. Vascular proliferation stage: Extension into the vitreous.
4. Retinal detachment.

It has been generally recommended that oxygen concentration in the case of premature infants should not exceed 40%, and then only for as brief periods as possible.

B—*Retinopathy of Diabetes Mellitus:* Retinal changes present the most serious problem confronting the ophthalmologist as far as the diabetic patient is concerned. Diabetic retinopathy is considered to be a specific clinical entity more closely related to the duration than to the severity of this metabolic disturbance. There appears to be a close relationship between the development of severe retinal lesions and renal involvment, reduction of renal function, intercapillary glomerulosclerosis, and secondary hyperfunction.[10]

Characteristic diabetic changes are usually bilateral and are found chiefly between the upper and lower temporal vessels and in the area about the optic nerve. The disk is always normal in appearance. The veins frequently are distended. Small round micro-aneurysms and punctate hemorrhages are noted along the course of the superior and inferior temporal veins. Visual disturbances depend on the degree of involvement of the macular area.

The most important part of the treatment and prevention of diabetic retinopathy lies in the degree of control of the diabetes itself. Joslin has shown that strict regulation of diabetes (blood sugar kept below 125 mgm per 100cc) lowers the incidence and severity of diabetic retino-

pathy. The value of medications such as rutin, ascorbic acid, hesperiden, vitamin B complex, and testosterone is highly questionable. Recently, vitamin B-12 deficiency has been found to be associated with excessive adrenal cortical function in diabetics.

C—*Hypertensive Disease:* In hypertensive retinopathy, one should attempt to differentiate the following significant fundus findings:

1. Angiospasm vs. permanent sclerotic changes (of particular importance in the toxemia of pregnancy).
2. Vascular changes of the senile nonprogressive vs. the progressive type.
3. Evidence of retinopathy; i.e. hemorrhages or exudates.
4. Evidence of malignant hypertension suggested by cotton-wool exudates, star-shaped figure of the macula, and papilledema.

Keith-Wagener's classification of hypertension is still the best working basis for determination of the degree of clinical activity:

Grade I—Local or generalized arteriolar attenuation.

Grade II—Advancing attenuation (the copper-wire reflex changing to the fine silver-wire appearance).

Grade III—Retinopathy (hemorrhages and exudates), silver-wire arterioles.

Grade IV—Papilledema, star-shaped figure, and cotton-wool exudates.

Long-continued hypertension and renal insufficiency are viciously inter-related.[11] Hypertensive fundi appearing with advanced arteriolar disease have an adverse prognostic significance. Here we may be entering the malignant phase of long standing essential hypertension or the end stages of a chronic glonerulonephritis.

D—*Ocular Manifestation of Collagen Diseases:* Klemperer, in 1942, first used this term to stress the importance of "systemic diseases of the connective tissue". In the ensuing years, the concept of a hypersensitivity response has been assumed.[12]

The systemic diseases most frequently included are:

1. Lupus erythematosus disseminatus
2. Periarteritis nodosa
3. Dermatomyositis
4. Generalized sclerodema
5. Serum sickness
6. Rheumatic fever
7. Rheumatoid arthritis.

Specific Ocular Changes: In lupus erythematosus disseminatus, periarteritis nodosa, dermatomyositis, and serum sickness we find the following early fundal changes at the posterior pole: (1) flame-shaped hemorrhages, (2) cotton-wool exudates, and (3) occasionally slight papilledema. These changes do not occur in rheumatic fever or rheumatoid arthritis. However, here we may find severe episcleritis, nodular scleritis and iridocyclitis. A nongranulomatous iritis, retinal perivasculitis, and occlusion of the central retinal artery have been found in periarteritis nodosa. The cardinal factor in pathogenesis appear to be damage to the capillary endothelium.

Therapy: Nonspecific therapy (i.e. the steroids: hydrocortisone, cortisone, prednisone, and prednisolone) has received the most attention in attempting to prevent the development of fibrinoid reactions or inducing their reversal after occurence.[13] Specific forms of therapy such as anticoagulants, gold therapy and nonspecific measures are being tried in treating the individual diseases.[14]

E—*Retinal Vascular Occlusions:* Anticoagulant therapy, vasodilators, and stellate-ganglion block are chief forms of therapy most commonly used in arterial and venous occlusions. Arterial occlusions frequently are accompanied by an element of spasm, and respond best when treated within the first 24 hours. In general, better results are obtained in the venous occlusions even where treatment has been started within the first few weeks following the advent of the occlusion. Anti-coagulants have proved most helpful in the venous occlusions where prothrombin levels may frequently be found above normal.[10]

F—*The Retina in Systemic Diseases:*

1. *Toxoplasmosis:* This disease has become an increasingly important cause of uveitis and chorio-retinitis. It appears both in congenital and adult forms. Diagnosis depends on a positive Sabin-dye-test titer, complement-fixation, and skin tests. Daraprim and the sulfonamides are the latest accepted forms of therapy.
2. *Tuberculosis of the Retina:* Isoniazid,[15] streptomycin, and aminosalicylic acid in combination is the best therapy for ocular tuberculosis. Sustained chemotherapy is necessary. Periphlebitis and vitreous hemorrhages are complications of fundal involvement.

G—*Blood Dyscrasias:* Hematologic diseases produce characteristic fundus changes: i.e. change in the fundus color and vascular caliber, hemorrhages and exudates, and, at times, edema of the optic disks. Evidence of these changes points to a blood dyscrasia.[16]

1. *Anemia:* Retinal hemorrhages usually appear when hemoglobin concentration drops to 35% or 5 Gm. per 100cc of blood. These hemorrhages are most likely secondary to increased capillary permeability.
2. *Polycythemia:* Cyanosis retinae, darkening of the retina, may result from primary or secondary polycythemia. Changes generally appear when the red cell count reaches 8,000,000 or the hemoglobin concentration reaches 115%. Hemorrhages are uncommon, but vascular dilitation may be found, especially in the veins. If papilledema occurs, the possibility of a tumor of the posterior fossa should be kept in mind.
3. *Leukemia:* Retinal hemorrhages often occur in all types of leukemia. They are more frequent in acute cases and in myelogenous leukemia than in lymphatic leukemia. The white-centered hemorrhage is the characteristic lesion. This probably results from leukocytic conglomerations surrounded by blood. Papilledema may occur and is more often associated with leukemia than with other blood dyscrasias.
4. *Thrombocytopenia:* Purpuric hemorrhages occur in the eye in essential and secondary thrombocytopenia. Small white exudates, probably ischemic infarcts of the retina are found in thrombotic thrombocytopenic purpura.

H—*Retinal Detachment:* The patient with a retinal detachment usually complains of a curtain-like loss of vision. Ophthalmoscopic examination reveals a retinal detachment corresponding to the area of the visual field loss. The patient may give a history of a minor blow to the eye or head but many retinal detachments are idiopathic or related to advancing myopia.

Diagnosis: This requires (1) thorough ophthalmoscopic examination with a widely dilated pupil, (2) transillumination thru the lids or sclera and noting of the red reflex through the pupil, (3) vision and visual field examination to corroborate the loss of vision and the scotomatous areas which should correspond to the detachment.[17]

Treatment: This requires early bed rest and surgery. Prognostically, Okamura and Schepens state that in cases of a retinal break with a minimum detachment, the probability of cure by operation is as high as 95%. If the detachment is allowed to progress, this figure will drop to 50% or 60%, owing to the formation of fixed folds in the retina plus vitreous retraction. Early cases with minimum detachment are chiefly treated by diathermic surgery. The more advanced cases are now treated by scleral buckling whereby the choroid is folded inward and brought into contact with the detached retina. Schaffer has recently introduced the idea of vitreous implantation, whereby the detached retina is pushed back against the choroid and diathermy alone or with scleral buckling is also performed.

I—*Tumors of the Retina:* So-called "gliomas" or neuroepitheleomas of the retina are usually detected before the age of four.[18] In this condition the mother usually brings the child because she has

noted: (1) a white "cat's eye" pupil, (2) evidence of poor vision, or (3) squint. Family history may reveal a strong hereditary tendency. Examination reveals a whitish retinal detachment which may fill the entire posterior segment to the back of the lens. At least 25% occur bilaterally. Diagnosis may be aided by P-32 radio-active studies.

Treatment: Most ophthalmologists feel that this malignant growth is best removed by early enucleation of the involved eye. If the second eye is involved, irradiation of the tumor and preservation of some vision can be attempted, but one is confronted with the possibility of metastasis and intracranial extension.

IX. NEURO-OPHTHALMOLOGY

The close relationship of the eye to the brain predisposes it to certain diseases of the nervous system. Prompt diagnosis is of utmost importance if blindness is to be prevented in those diseases for which there is effective treatment. Among the important ocular manifestations of neurologic diseases are: (1) ptosis; (2) lagophthalmos (inability to close the eye); (3) abnormal extraocular mitility; (4) loss of corneal sensitivity; (5) pupillary changes as to size and reactivity; (6) visual loss due to central scotoma; (7) papilledema and atrophy of the optic nerve; (8) and contractions or scotomas in the visual fields.[19]

Today, the ophthalmologist must be alert to the possibility of disorders of the nervous system that may damage vision, in order that the patient may be referred to the neurosurgeon early so that a cure may be affected.

A—*Eye Movement Disturbances and Palsies:* The nerves to the eye muscles take a long and vulnerable course along the base of the skull and orbit. Roentgenologic examination should include views of the skull, special views of the orbits, paranasal sinuses, petrous bones and base of the skull. Also, where indicated, roentgenologic contrast methods (i.e., angiography and pneumography) are used.[20]

Ocular Palsies: Gross lesions causing paralysis of the extraocular muscles are usually located in the region of the third, fourth, and sixth cranial nerves in the cranial cavity and only rarely in the orbit.[21]

1. *Third Nerve:* Paralysis of the third nerve required surgical correction; and is most commonly caused by intracarotid aneurysm. Typical signs are ptosis, diplopia, and third nerve palsy.
2. *Fourth Nerve:* Trochlear paralysis affects the superior oblique muscle, the depressor of the globe. Head trauma and vascular diseases are the most common disturbances; tumors and aneurysms are rare.
3. *Sixth Nerve:* Paralysis of the abducens is the most common of all extraocular palsies. Due to its long intracranial course it is vulnerable to mechanical compression, adjacent inflammatory, or vascular disease. Sixth nerve involvement is common with brain tumors, but is of little localizing value per se.[22]

B—*Supranuclear Lesions:* Lesions here are characterized by an inability to perform conjugate eye movements in one direction, either in horizontal or vertical planes. Diplopia is absent because the deviation is equal in both eyes.

C—*Intranuclear Ophthalmoplegias:* This occurs when there is a lesion of the medial longetudinal fasciculus. The characteristic feature of this condition is paresis of one medial rectus; or more frequently, both, on attempted lateral gaze. Convergence is usually intact, particularly in the more common posterior internuclear ophthalmoplegia. It may be diminished or absent in anterior internuclear ophthalmoplegia; that is, the midbrain portion.

D—*Intracranial Surgical Lesions Affecting Visual Acuity:* These are gross space-occupying lesions inside the cranial cavity closely surrounding the optic nerves and chiasm or, less often, encroaching on the visual tracts or the occipital visual centers. Practically all of these lesions are benign and can be removed safely and successfully when symptoms first arise.

Diagnostic features are: (1) slowly diminishing vision, (2) progressive visual field cuts, (3) optic atrophy, and (4) choked disks. Although plain x-rays of the skull may be normal, intracranial air injection and arteriographic studies usually will reveal the true nature of the eye disorder. The commonest causes are: (1) aneurysms, (2) hypophyseal adenomas, (3) Rathke tumors, (4) meningiomas, and (5) osteomas.[23]

E—*Lesions of the Cerebellar System:* The cerebellum serves as a coordinator and modulator of extraocular innervation. In extensive cerebellar disease, eye movement disturbances occur that are analagous to those of the extremities, i.e. asynergia, ataxia, and dysdiadokokinesis of eye movements. Here we find disassociation of conjugate movements of both eyes.

F—*Pituitary Tumors:* These are one of the most frequent neurologic conditions producing loss of vision. Dimness of vision in one eye is soon followed by difficulty with the other eye. Reduced visual acuity is not corrected by glasses and this calls for an immediate plotting of the visual fields. This alone may determine evidence of a pituitary tumor. Pallor of the optic nerve head from pressure plus enlargement of the pituitary fossa may also be found if the disease is discovered at a later stage.

G—*Meningiomas:* This large group of tumors are only malignant in their pressure effects on adjacent structures. Due to their proximity to the optic nerve, usually one eye is affected at first. Loss of vision is slow and painless. Field loss is not as diagnostic as in pituitary tumors but x-rays aid in the diagnosis in 50% of the patients.

H—*Cerebrovascular Disease:* The usual "stroke", when it affects vision, blacks out a half of the patient's visual field, a homonymous hemianopsia. If a vascular accident involves a basilar artery which supplies the occipital lobes of the brain and the visual centers, the patient may be completely or nearly blind. Transient blacking out of vision sometimes occurs in some patients and should be treated as a forewarning of impending disaster.[24]

I—*Multiple Sclerosis:* These patients frequently develop optic neuritis with slight discomfort in and about the eye, followed by sudden blindness (one or two days). Fortunately, the vision usually improves in four to six weeks and may return to normal. If repeated attacks occur, permanent reduction of visual acuity results. According to Duke-Elder, the combination of uniocular nystagmus and impairment of lateral conjugate gaze is pathognomonic of multiple sclerosis. Each medial rectus is impaired but convergence is normal.

X. VISUAL AIDS

A—*Contact Lenses:* Despite their expense, contact lenses are being increasingly used for a greater number of ocular conditions by a more educated public. Fitting of these lenses has been simplified since the adoption of the smaller microtype corneal lenses which are used without special fluids.[25]

Indications—In brief, contact lenses are useful in the following conditions:

1. Keratoconus and irregular astigmatism due to corneal scarring.
2. Unilateral aphakia, thereby producing binocular vision by reducing the size of the image. (Also now used in binocular aphakia for wider field and cosmetic improvement).
3. For cosmetic reasons, and for athletics where glasses cannot be worn.
4. Corneal protection against exposure keratitis in facial paralysis.

B—*Visual Aids for Subnormal Vision:* Of the more than 300,000 legally blind in the United States 60% have a little sight. Impaired vision has been roughly classified as follows[26]:

1. Vision of 4/200 equals social blindness.
2. Vision of 20/200 or less in the better eye equals occupational or legal blindness.
3. Partially seeing group equals vision below 20/70. In the classroom, these children need special educational help. For those who recognize hand

movements only, no optical aid is helpful. An optimistic outlook is warranted, however, if a person can read large headlines with his ordinary correction. With optical aids the partially sighted frequently can read and continue in occupations when they otherwise could not.

Various telescopic magnifiers are now available, giving magnification at distance of 1.8 times up to 2.2 times. A plus 4.00 reading lens may be placed over this lens for near vision, thereby increasing the size of the image sufficiently to be legible. Greater magnification for near vision can be obtained but the field of vision and working distance are correspondingly reduced.[27]

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THE ARTERY BANK OF DELAWARE

WALTER W. MOORE, M.D.

Surgeons in the various hospitals of Delaware have realized for the past several years that there is a need in our community for a suitable material to be used to replace segments of arteries damaged in accidents, as well as those damaged by disease, such as aneurism. To accomplish this end, many substances such as Orlon, Nylon, and Dacron, have been tested and there are sporatic reports of success using many different materials. However, experience at the present time would indicate that the best material available for arterial grafting is the natural freeze-dried arterial homograft. This is especially true in cases where smaller vessels are in need of repair.

Many problems have arisen in the preparation of adequate freeze-dried arterial homografts. The first problem was that of obtaining permission from the immediate relatives of the deceased patient to remove segments of arteries for preparation and storage. At first this appeared to be an unsurmountable stumbling block; however, experience has shown that by the proper approach to the responsible people concerned over 50% will give immediate permission in writing for removal of such tissues at autopsy. No tissue is removed until full permission (signed and witnessed) has been granted.

Our second problem was that of obtaining sterile vessels. During the early part of our work, a sterile autopsy was performed in each case; however, it was soon discovered that many tissues taken from the body by a "sterile autopsy" would be grossly contaminated by colon bacilli if they were taken later than twelve hours after death. Obviously, we were forced to discard many tissues after we discovered that they had been contaminated so that only 10% of the available tissues could actually be used. It also became apparent that some different process would have to be made available to us for the sterilization of these vessels so that the ones which were contaminated would not have to be destroyed. This problem was answered by the discovery of the beta-propiolactone technique[1] which made it possible to sterilize blood vessels without damaging the tissue itself. This was done in the following manner. A freshly prepared 1% solution of beta-propiolactone (BPL) is placed in a suitable wide-mouthed flask containing the vessel to be sterilized. The flask containing the artery and the BPL is then placed in a water bath at 37° C. for two hours. During this time, the BPL hydrolizes and destroys any organisms on the vessel thus making it unnecessary to do a sterile autopsy. After this process has been carried out, cultures are taken, both of the solution and of the vessel surface, before the vessel is freeze-dried. In no instance have we encountered any contamination of the tissue after it has been sterilized by the beta-propiolactone technique.

The artery thus prepared is now ready for freeze-drying so that it can be stored at room temperature. To accomplish the freeze-drying, the artery is removed aseptically from the bicarbonate solution and placed into a sterile long-necked tube. The tube containing the artery is then plunged into a solution of acetone and dry ice at a temperature of —76° C. This low temperature quickly freezes the vessel, thus causing the formation of very tiny crystals rather than large crystals which would form if the vessel were frozen slowly. The formation of small crystals is essential if one is to prevent deterioration of the tissue due to freezing. This tube containing the frozen artery is then placed on the freeze-drier, described in Figure 1, the vacuum pump is put into action, and a vacuum is drawn on the apparatus. As the atmospheric pressure within the system diminishes to approxi-

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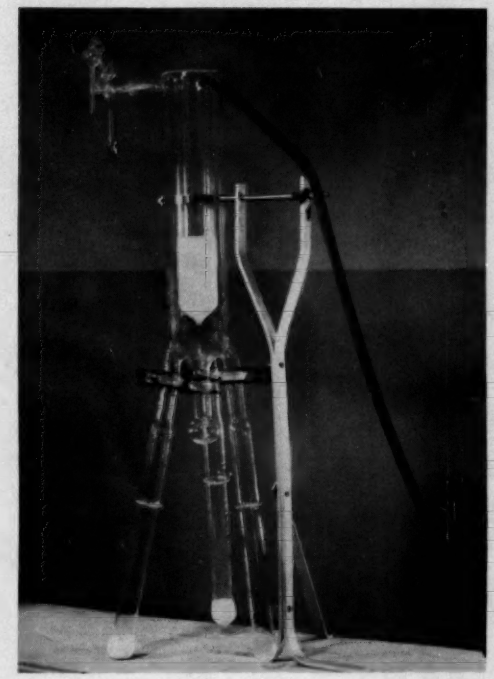


FIGURE 1

Note the freezing solution of acetone and dry ice in the inner chamber, the vacuum gauge on the upper left, the line to vacuum pump on the upper right, and the arteries being dried in the three lower tubes.

mately 15 microns, the kinetic energy of the molecule of the water on the surface of the artery enables the water to pass from the solid to the gaseous stage (sublimination) only to be recondensed on the cold surface of the condenser, which contains a solution of acetone and dry ice with a temperature of —76° C. As the process continues, all the moisture leaves the artery to be recondensed on the surface of the condenser. After approximately eighteen hours all the moisture has been removed from the vessel and it appears white and dry. A large frost appears on the condenser. At this time the vacuum is released, the vacuum pump is turned off, and the glass tubes are removed from the freeze-drier. These tubes are then taken to a glass blowers laboratory where they are immediately sealed under a high vacuum, using the glass blowers torch. A flange is also placed on the neck of the glass tube, as seen in Figure 2. This flange will enable one to crack the glass container at that point with the slightest twist. After vacuum sealing, the dried artery can be stored at room temperature to be used at any time.

When the patient arrives in the operating room with an apparent arterial injury requiring an artery graft, the sterile tube containing the sterile freeze-dried artery is placed on the operating room table. The tube is broken and the vessel is reconstituted by simply bathing it in sterile saline solution for twenty minutes before use. The vessel thus regains the soft, pliable texture that it had previous to freeze-drying, and is now ready for implantation (end to end or end to side anstomosis) to the damaged artery.

The technique of vascular anstomosis is not a difficult one if one remembers a few simple technical rules. First, debridement must be adequate so that there will be no necrotic tissue present to help cause wound infection and graft failure. Second, the ends of the damaged artery must be freed of frayed edges and hematoma so that a close approximation of the tissue with the arterial graft can be accomplished. Third,

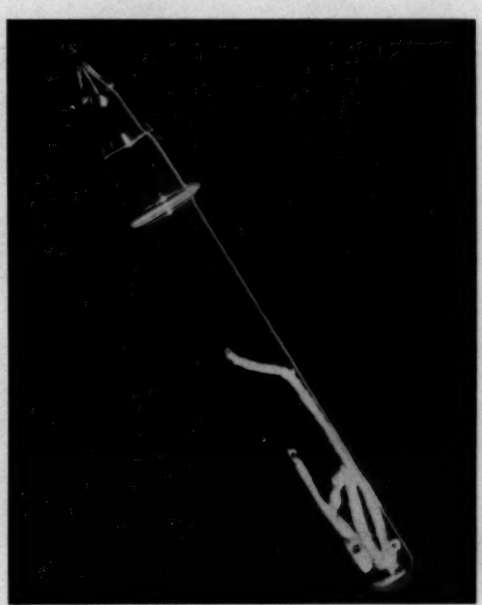


FIGURE 2

Freeze-dried femoral and popliteal arteries sealed in a vacuum tube ready for storage at room temperature.

tension between the severed ends of the artery and the graft itself must be minimal. Fourth, the suture which is 4-0 Dekknettkal in the larger vessels and 5-0 Dekknettkal in the smaller vessels should be placed as a loose, not tight, continuous through and through everting suture around the entire vessel. The suture is placed loosely so that as the arterial clamps are released and the blood begins to flow through the vessel, the vessel will begin to stretch and the suture will become tighter. As the vessel assumes its normal size the suture will become tight and all bleeding will cease. If, however, the suture is placed tightly before the blood is allowed to flow, stenosis will form at the site of the anstomosis when the vessel is stretched to its normal size. This will cause thrombosis and graft failure. In repairing larger vessels such as the aorta, I have found it satisfactory to sew the aorta in four quadrants starting posteriorly. A continuous suture is started at six o'clock and carried up to the nine o'clock position where it is tied to a second suture which is started at nine o'clock and carried to the twelve o'clock position. This in turn is tied to a suture which is started at twelve o'clock and carried to the three o'clock position, and so forth. After an adequate blood flow is established through the vessel, the wound is irrigated, muscle flaps are placed over the vessel, and the skin is closed, either by direct suturing or pedical grafting if necessary. It is essential that these patients are given antibiotic therapy, and that the afflicted limb be immobilized in a plaster cast. Failure of immobilization can be one of the prime causes of graft failure in traumatic cases.

As tissues become available and are preserved in this manner, they are being stored in complete sets in the operating rooms of the various hospitals to be available in any emergency. Once each hospital in Delaware has secured an adequate supply, the Central Artery Bank will stockpile the additional arteries and make them available to the hospitals as the need arises.

The author acknowledges the technical assistance of Mr. Robert Varland and Mr. Harry Weir of the Atlas Powder Company.

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THE PHYSIOLOGICAL ACTION OF THE TRANQUILIZER DRUGS

by

K. M. CORRIN, M.D.*

Probably the greatest benefit of the tranquilizer drugs has been to bring about in the mind of physicians the fact that disorders of the brain and nervous system are essentially organic in nature, the same as physiological and pathological disorders of any other organ, and that they respond in the same way to drug therapy. Diagnosis of these disorders is also becoming more and more accurate—anatomical and physiological in nature, rather than symptomatic, as in the past. This change in thinking calls for a more specific type of drug therapy and one finds in the tranquilizer drugs, agents which tend to have a major effect on specific parts of the nervous system, just as symptoms point to disorders in specific parts. The following briefly summarizes these major physiologic effects of the basic tranquilizer drugs.

Chlorpromazine (Thorazine), and its less potent cousin *Compazine*, act primarily on cortical functioning, blocking ideation impulses—fears, illusions, delusions and hallucinations. This drug was early considered a chemical substitute for lobotomy and is still used as such. In the borderline psychotic its skillful use, in combination with related therapy, may keep the patient out of a mental institution.

Rauwolfia (Reserpine) apparently acts in modifying nerve impulses from the midbrain and brain stem structures. Instinctive tension, fears and worries, as seen in the agitated senile individual, and the patient with hypertension of nervous origin, often respond well to Reserpine.

The *belladonna alkaloids* (atropine especially) are the best drugs to quiet autonomic overstimulation—the result of overaction of the post ganglionic (cholinergic) visceral efferent fibres. Overstimulation of this system is the chief factor in producing the troublesome symptoms of the neuroses—chiefly visceral and sphincter tension and spasms, relatively chronic in nature. Given in small doses, alone or in combination with phenylethyl barbituric acid (phenobarbital), and over a long period of time, the doctor will often find it to be his most effective and satisfactory drug in treating nervous tension states.

Meprobamate (Miltown, Equanil) acts somewhat like curare, blocking nerve impulse transmission at neuromyal junctions and to a lesser degree at autonomic ganglionic synapses. It may often be combined effectively with belladonna to quiet both the somatic and visceral musculature.

Phenylethyl barbituric acid (phenobarbital) is probably the most commonly used, the safest, the cheapest and the most effective of all the tranquilizer drugs. In addition to its being the best and safest anticonvulsant agent for long term use, it is also an excellent hypnotic when given in proper dosage. Its physiological action is that of a central nervous system depressant, the degree depending on the dosage—in small doses it acts as a tranquilizer, in moderate doses a hypnotic, and in large doses a depressant.

In the use of these drugs, and the treatment of nervous and mental disorders generally, there are several essentials which the physician should keep in mind.

First, that these drugs are potent and complete knowledge of their actions and

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effects are necessary for their most effective use.

Second, that close medical supervision is required—they are not for self medication.

Third, that optimism, patience and time are essential in the treatment of all nervous and mental disorders.

Fourth, that often, when one drug or dosage is unsatisfactory, a change to another drug or combination may be more effective.

Fifth, that confidence of the patient in the therapist and his ability is part of the treatment. This is best obtained by the doctor taking time to obtain an unhurried history including a little of the personal side of the patient's life (hopes, fears and behind-the-scene problems), plus a good physical examination and laboratory study. A little time-taking psychotherapy is uniformly necessary in the treatment of all nervous and mental patients but the family doctor with a medical outlook, who will take a little extra time for this psychotherapy, will always have the patient's confidence to a greater degree than the psychiatrist with a purely psychological point of view. This is essential in influencing the patient to follow suggestions and continue treatment until recovered.

PICKWICKIAN SYNDROME*

ANTERO Q. SORIANO, M.D.**
AND
J. R. DURHAM, JR., M.D.***

There is a rare association of signs and symptoms described by Charles Dickens in "The Pickwick Papers". His description of the fat boy, a character with marked obesity and somnolence, has given rise to the term "Pickwickian Syndrome". Its clinical features are marked obesity, somnolence, twitching, cyanosis, periodic respirations, secondary polycythemia, right ventricular hypertrophy and failure.

Review of the recent publications on this subject[1] are of particular interest in the case to be presented. Several writers on this syndrome have indicated that increasing abdominal girth is accompanied by a decreasing expiratory reserve and if the increase in girth is extreme, it may lead to a state of relative ventilatory insufficiency.

Sieker and his colleagues[3] in 1954 have reported four patients with extreme obesity and in whom somnolence, periodic respirations, intermittent cyanosis, polycythemia and right axis deviation were noted. Ventilatory studies revealed diminished total lung volume, diminished expiratory reserve volume and diminished functional residual capacity. The study made by Sieker and others is the first modern and scientific description of this syndrome which they named "obesity heart disease".

Auchincloss and others[2] stated that these patients with obesity, cyanosis, dyspnea and edema have erythrocytosis and right ventricular hypertrophy but no evidence of disease of the lungs, chest wall or central nervous system. Physiologic study showed the presence of marked arterial hypoxia, hypercapnia and severe alveolar hypoventilation. This was characterized by a marked reduction in tidal volume without an increase in respiratory frequency sufficient to maintain normal alveolar hypoventilation. Marked pulmonary arterial hypertension and increased cardiac output were also present. There was no evidence that these findings were initiated by an abnormal cardiac or vascular communication. Treatment with bed rest, low salt diet, mercurials, digitalis and phlebotomy was associated with disappearance of dyspnea and edema. When studied after ten months of such treatment, arterial hypoxia and hypercapnia and alveolar hypoventilation were still present, although to a lesser degree than at the time of initial observation. All of the physiologic abnormalities have been attributed to the alveolar hypoventilation. However, the initiating mechanism for the alveolar hypoventilation was not discovered.

CASE REPORT

A 35-year-old white, married carpenter was admitted to the Wilmington General Hospital on November 19, 1956 with the chief complaint of being unable to stay awake. For more than a year, he frequently lapsed into sleep at unpredictable times, so much so that he would fall asleep at the table, while talking and even while at work. These sleeping episodes were intermittent. He usually slept for two to five hours duration. Sometimes, he was extremely difficult to awaken and would sleep one to two days if not disturbed. During the past year, he had attacks at the rate of one to two a week. He had gained weight gradually for the last four to five years and one month prior to admission his weight was 305 pounds.

* From the Medical Service, Wilmington General Hospital
** Chief Resident
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The past history revealed measles, mumps and chickenpox during childhood. One year previously he was admitted to another hospital with the chief complaint of "too much water in me". He was then in a state of somnolence. Laboratory studies showed red blood cells, white blood cells, hemoglobin, blood urea nitrogen, carbon dioxide combining power, blood sugar, total protein, and albumin/globulin ratio to be normal. There was one plus albumin in the urine. The blood serology was negative. The heart was top normal in size by x-ray and there was slight haziness in the left lung field. Two electrocardiograms revealed T-wave changes possibly due to digitalis effect, coronary insufficiency or subendocardial infarction. His blood pressure was 130/90. Treatment consisted of vitamins, diuretics, antibiotics and anticoagulants. His respirations became irregular and labored. He was aroused with great difficulty. After one week he walked out of the hospital against advice.

Three months before admission he was treated for cardiac failure and obesity with diuretics, digitalis and sedatives. He responded well to the regimen for awhile but then went into coma with asthmatic breathing; later on consciousness was regained.

The father was a tall, husky man who weighed 220 pounds and died as a result of an accident. The mother, brothers and sisters are living and apparently healthy and well. None of them is obese.

On admission to the Wilmington General Hospital he was markedly obese, cyanotic and so drowsy that he could not stay awake for a minute. He would respond to painful stimulation but became hostile and violent and struggled to get out of bed when forcibly awakened. Respirations were 26 per minute and irregular with loud snoring.

He was afebrile, his pulse was 90 per minute and his blood pressure was 136/90. There was nothing remarkable in the physical findings except for distant heart and breath sounds. The neurosurgeon who saw him thought that the cerebral manifestations were more compatible with polycythemia than with intrinsic intracranial disease.

The only laboratory studies which could be accomplished prior to death were: red blood cells 5,080,000, white blood cells 11,750, hemoglobin 14.8 Gm, segmented neutrophiles 79%, lymphocytes 13%, monocytes 8% and 3 nucleated red cells. Urinalysis showed specific gravity of 1.025, with sugar and acetone negative; however it was loaded with hyaline and fine granular casts. The carbon dioxide combining power was 64 volumes %, the blood sugar was 150 mg.%, blood urea nitrogen was 42 mg.% and the serology was negative.

He was put at complete bed rest and was given oxygen therapy and intravenous fluids. In spite of treatment, he remained cyanotic and continued to be comatose until he died fourteen hours after admission.

Autopsy resulted in the following pathological diagnosis: 1)—hypertrophy and dilation of the right ventricle; the heart weighed 850 Gm. 2) hydropericardium (100 c.c. of fluid) 3) bilateral hydrothorax 4) congestion of the lungs 5) chronic passive congestion of the liver 6) hepatomegaly 7) ascites 8) obesity.

SUMMARY

A short resume of the literature on the Pickwickian Syndrome with a case report is presented. The presence of alveolar hypoventilation in connection with this symptom complex has been discussed.

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Editorials

THE SLEEPY FAT BOY

It is altogether fitting that a new disease entity be called "The Pickwickian Syndrome" despite a popular aversion to the use of medical eponyms. While truly a controversial figure, Charles Dickens was acknowledged by all to be a keen observer and reporter of people. It therefore is not surprising to find in his writings some classical descriptions of disease.

Dickens was a contemporary of some giants of British medical history—Astley Cooper, John Abernethy, Richard Bright, Matthew Baillie, William Jenner, Dominic Corrigan and Joseph Lister to name but a few. It therefore is of unusual significance that in 1870 the editor of the British Medical Journal said that Dickens would have made a great physican because of his keen powers of observation.

The editor mentioned that Dickens' description of "hectic" had been incorporated into several standard textbooks and gave him credit for priority in describing the relationship of right hemiplegia to aphasia.

This Journal is proud, therefore, to present a case report of the Pickwickian syndrome and to hope that this name may survive.

ORAL ANTI-DIABETIC DRUGS

The Diabetes Committee of the Medical Society of Delaware wishes to warn against injudicious use of Tolbutamide (Orinase), a sulfonyl urea, recently released for sale upon prescription through regular drug channels.

Even in those patients whose blood and urine sugars are well controlled by Orinase, should an intercurrent infection arise, insulin must be used immediately to avoid acidosis and coma.

Orinase is ineffective in a juvenile diabetic.

Orinase should not be given to patients with liver disease.

Orinase may cause pseudo-albuminuria which may require complicated chemical tests to differentiate from true proteinuria.

This drug is not an insulin substitute; it is a hypoglycemic agent, the exact mode of action or long term effect of which is not yet known.

The Committee urges careful observation of patients who receive Orinase and will appreciate receiving information concerning any difficulties encountered.

BOOK REVIEWS

CURRENT THERAPY 1957: Latest Approved Methods of Treatment for the Practicing Physician. Editor: Howard F. Conn, M.D. Consulting Editors: George E. Burch; M. Edward Davis; Vincent J. Derbes; Garfield G. Duncan; Hugh J. Jewett; Perrin H. Long; Clarence S. Livingood; H. Houston Merritt; Walter L. Palmer; Hobart A. Reimann; Cyrus C. Sturgis; Robert H. Williams. 731 pages. 1957. W. B. Saunders Company, Philadelphia. $11.00.

This volume, the ninth in the series, follows a format established in the earlier editions. It is a collection of short essays on treatment, written by approximately three hundred contributors. The book is divided into sixteen sections, fifteen covering various phases of medicine and the sixteenth containing appendices and indices. The individual articles vary in quality and there is a noticeable difference in editing when the various sections are compared. The sections concerned with diseases of the respiratory system; disorders of metabolism and nutrition; diseases of the locomotor system; and diseases due to physical and chemical agents are well worth reading. Excellent tables listing active poisonous ingredients in various commercial products are available.

The major points of criticism are, first, that references are few and a bibliography is not available, making it difficult to check source material; second, controversial subjects are covered without debate. In this reviewers opinion any book which is used for reference must at least recognize differences of opinion. While it is an author's prerogative to stress his own views, it is also incumbent upon him to at least mention other recognized ideas.

This book is current therapy only for each individual author. It is not necessarily the best or only therapy applicable in the diseases discussed. For these reasons this book is recommended only for those already familiar with the diseases and therapies covered.

L. P. L.

EPILEPSY AND THE LAW. By Roscoe L. Barrow, Dean, University of Cincinati College of Law; Legal Advisor, Special Committee on Legislation, American League Against Epilepsy; and Howard D. Fabing, M.D., Past President, American Academy of Neurology; Chairman, Legislation Committee, American League Against Epilepsy. 177 pages. 1956. Paul B. Hoeber, Inc., New York. $5.50.

"Epilepsy and the Law" is a proposal for legal resume' in the light of medical progress in the diagnosis, therapy and rehabilitation of epileptics. The authors have combined their knowledge and background to review the current status of the legal aspects of epilepsy and have made valuable suggestions as to future alterations of a more realistic nature. The book consists of an introduction stating the facts of epileptic frequency and the success in its therapy and then goes on to discuss the specific recommendations in the field of (1) marriage license laws, (2) sterilization laws, (3) driver's license laws, and (4) workmen's compensation laws.

The book is endorsed by the special committee on legislation of the American League Against Epilepsy. Of the approximately one and one-half million epileptics in the United States, almost 80% have their seizures satisfactorily controlled by proper medication. Yet despite this, society has been prejudiced against the epileptic so that he suffers socially, economically and psychologically. It is to remove the stigma of isolation and discrimination which does not exist realistically in light of modern medical knowledge and progress that this book was written.

The current status of the marriage license laws pertaining to epileptics is shocking. The same situation applies to driver's license laws, and the book contains a model statute which already has been operating successfully in Wisconsin. It is felt that the current laws tend to drive epileptics "underground" and create far more hazards and difficulties than the help which they presume to afford. The remainder of the discussion applying to workmen's compensation laws and miscellaneous laws affecting epileptics such as immigration, criminal responsibility of the epileptic, commitment of epileptics to institutions, and education of epileptic children, is excellently written. The last section on findings and recommendations is concise and clear.

It is the hope of this reviewer that this book will be but the first of a series of

similar surveys and recommendations. Certainly there are many illnesses which have created about them an aura of prejudice and stigma with resulting socio-economic detriment to the individual. We hope that this book will point the way.

J. K.

CLINICAL USE OF RADIOISOTOPES. By William H. Beierwaltes, M.D., Associate Professor of Internal Medicine and Coordinator, Clinical Radioisotope Unit, University Hospital, Ann Arbor; Philip C. Johnson, M.D., Assistant Professor of Internal Medicine and Chief, Radioisotope Unit, Veterans Administration Hospital, Oklahoma Medical School, Oklahoma City; and Arthur J. Solari, B.S., M.S. (Physics), Instructor in Radiation Physics, Department of Radiology, Radiation Physicist for Clinical Radioisotope Unit and Kresge Research Iostope Unit, University Hospital, Ann Arbor. 456 pages with 126 illustrations. 1957. W. B. Saunders Company, Philadelphia. $11.50.

This book is a good survey of the clinical application of radioactive isotopes. It is especially suitable for persons who are actually engaged in the use of these tools or for those who hope to institute an isotope program. Many sections of the book such as the ones on thyroid physiology and on I-131 tests of thyroid function, are of value for any clinician who has patients needing isotope tests. Except for the sections on physics, atomic energy commission requirements, and instrumentation, the text is clinically centered and thus is understandable to those who have had no preliminary experience with radioactive medicine. While theoretically the novice could start from the beginning of the book and develop a good understanding of the theories and practices of an isotope laboratory, the book itself will not substitute for experience in the laboratory.

In an effort to make the physics clear, some of the explanations seem a bit brief. The problems of counting statistics and geometry (especially the latter) are not sufficiently stressed and I wished that the authors' experiences with drugs and medical conditions that alter iodine uptake had been further elaborated. One fascinating comment on the rebound phenomenon in a gland previously suppressed by thyroid administration is left undeveloped and "in the air". On the other hand, much personal experience is reviewed, with the authors' choice of methods from the many various tests available for using isotopes.

The bibliographies at the end of each chapter are extensive and thus of distinct value in a young science where developments are rapid and techniques are frequently not yet standardized.

On the whole the book is excellent. The minor criticisms do not detract from its overall value.

R. W. F.

MEDICINE'S CONTRIBUTION TO WORLD PEACE

Medicine is universally recognized as one of the great world-wide arts and sciences that bind humanity together with a language and a purpose transcending all differences of race, creed or color.

To make the language of medicine more articulate in the cause of international peace and human progress, the doctors of the free world are united in The World Medical Association, whose membership now embraces 53 national medical associations.

But it is never enough to establish great institutions. Only when *individuals* are given an opportunity to play an active part does any human organization "come alive" and begin to realize its basic purposes.

Every American doctor knows first hand the vital role he may play in guiding and protecting his profession by becoming an active member of his county, state and national medical societies.

Today, every American doctor has the opportunity—and the imperative challenge to help make our profession a stronger influence for world peace. This he may do by joining our own United States Committee of The World Medical Association.

Similar "supporting committees" have been organized in a number of other leading nations whose national medical societies, like the A.M.A., are members of W.M.A.

In a timely action, W.M.A., at its 10th General Assembly in Havana in October, adopted a six point program to implement one of its constitutional purposes: to promote world peace. This program includes the development of mutual exchange visits of foreign doctors; exchanges of distinguished medical teachers; establishment by each W.M.A. member national association of an "international visitor's bureau"; stimulation of visits by representatives of member associations to the annual meetings of other member associations; holiday exchange programs between doctors and their families; and exchanges of text books and medical and scientific publications.

To implement this program takes money — and interested members. YOU may play your part by joining the U. S. Committee of W.M.A. Active membership dues for 1957 are $10.00. To join the U. S. Committee — and to learn how you can contribute to this great cause — communicate with Dr. O. J. Pollak, 9 Kings Highway, Dover, Del.

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Seek a friend's advice, if you wish, on almost any other problem. But when it comes to your health, and that of your family, by all means don't let anyone other than a physician advise you.

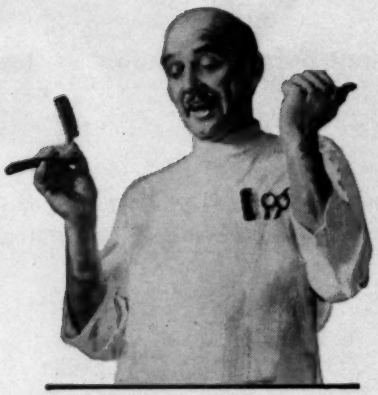
By seeing your doctor at the first sign of trouble, you will not only avoid the hazards of amateur medical advice, but chances are you will save time and money in the long run. In fact, prompt and proper medical care may well turn out to be one of the biggest bargains ever to come your way

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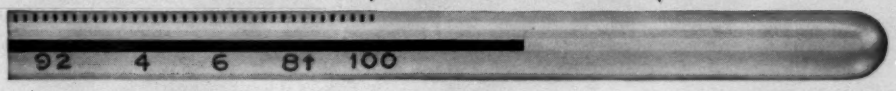
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(1) King, J. W., and Hainline, A., Jr.: Commercial Glucose Oxidase Preparations for the Detection of Glucose in Urine, Cleveland Clin. Quart. 23:212, 1956. (2) Leonards, J. R.: Evaluation of Enzyme Tests for Urinary Glucose, J.A.M.A. 163:260 (Jan. 26) 1957.

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(1) Holt, J. O. S., Jr.: Dallas Med. J. 42:497, 1956. (2) Gelvin, E. P.; McGavack, T. H., and Kenigsberg, S.: Am. J. Digest. Dis. 1:155, 1956. (3) Natenshon, A. L.: Am. Pract. & Digest Treat. 7:1456, 1956.

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*Income payable from first day of medical attention and as long as continuous total disability, total loss of time and medical attendance continue

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RUBRATON

SQUIBB IRON, B COMPLEX AND B_{12} VITAMINS ELIXIR

- to correct many common anemias
- to correct mild B complex deficiency states
- to aid in promotion of growth and stimulation of appetite in poorly nourished children

SQUIBB

Squibb Quality—
the Priceless Ingredient

Each teaspoonful (5 cc.) supplies:

Elemental Iron (as ferric ammonium citrate and colloidal iron) (equivalent to 130 mg. ferrous sulfate exsiccated)	38 mg.
Vitamin B_{12} activity concentrate	4 mcg.
Thiamine mononitrate	1.0 mg.
Riboflavin	1.0 mg.
Niacinamide	5 mg.
Pantothenic acid (Panthenol)	1.5 mg.
Pyridoxine hydrochloride	0.5 mg.

Alcohol content: 12 per cent

Dosage: 1 or 2 teaspoonfuls t.i.d.

Supply: Bottles of 8 ounces and 1 pint.

"RUBRATON"® IS A SQUIBB TRADEMARK.



GRADATIONS OF ANALGESIA

'TABLOID' 'EMPIRIN' COMPOUND®
Acetophenetidin gr. 2½, Acetylsalicylic Acid gr. 3½, Caffeine gr. ½

'TABLOID' 'EMPIRIN' COMPOUND with CODEINE PHOSPHATE gr. ⅛, No. 1 (N)

'TABLOID' 'EMPIRIN' COMPOUND with CODEINE PHOSPHATE gr. ¼, No. 2 (N)

'TABLOID' 'EMPIRIN' COMPOUND with CODEINE PHOSPHATE gr. ½, No. 3 (N)

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PAIN CONTROL
with sedation

GRADATIONS OF ANALGESIA
with light sedation

'EMPIRAL'®

Phenobarbital	gr. ¼
Acetophenetidin	gr. 2½
Acetylsalicylic Acid	gr. 3½

'CODEMPIRAL'® No. 2(N)

Codeine Phosphate	gr. ¼
Phenobarbital	gr. ¼
Acetophenetidin	gr. 2½
Acetylsalicylic Acid	gr. 3½

'CODEMPIRAL'® No. 3(N)

Codeine Phosphate	gr. ½
Phenobarbital	gr. ¼
Acetophenetidin	gr. 2½
Acetylsalicylic Acid	gr. 3½

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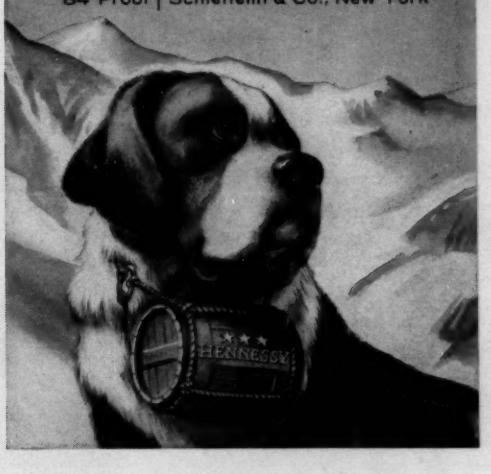
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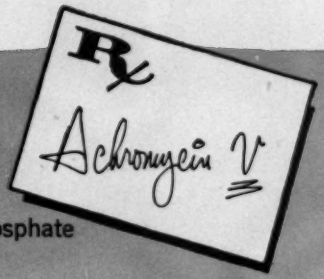
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V stands for— • **greater antibiotic absorption** • **earlier therapeutic blood levels** • **faster broad-spectrum action.** ACHROMYCIN V Capsules are the new, rapid-acting, oral form of ACHROMYCIN* Tetracycline—offering your patients, on the average, twice the antibiotic absorption in half the time required by older preparations.





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ACHROMYCIN® V Tetracycline Buffered with Phosphate

CAPSULES—Each capsule (pink) contains tetracycline equivalent to 250 mg. of tetracycline HCl, phosphate-buffered. Bottles of 16 and 100 capsules.

SYRUP—Each teaspoonful (5 cc.) of orange-flavored syrup contains 125 mg. of tetracycline HCl activity, phosphate-buffered. Bottles of 2 and 16 fl. oz.

ACHROMYCIN V dosage: 6-7 mg. per lb. of body weight per day for children and adults.

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WHO SUFFERS
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longest acting
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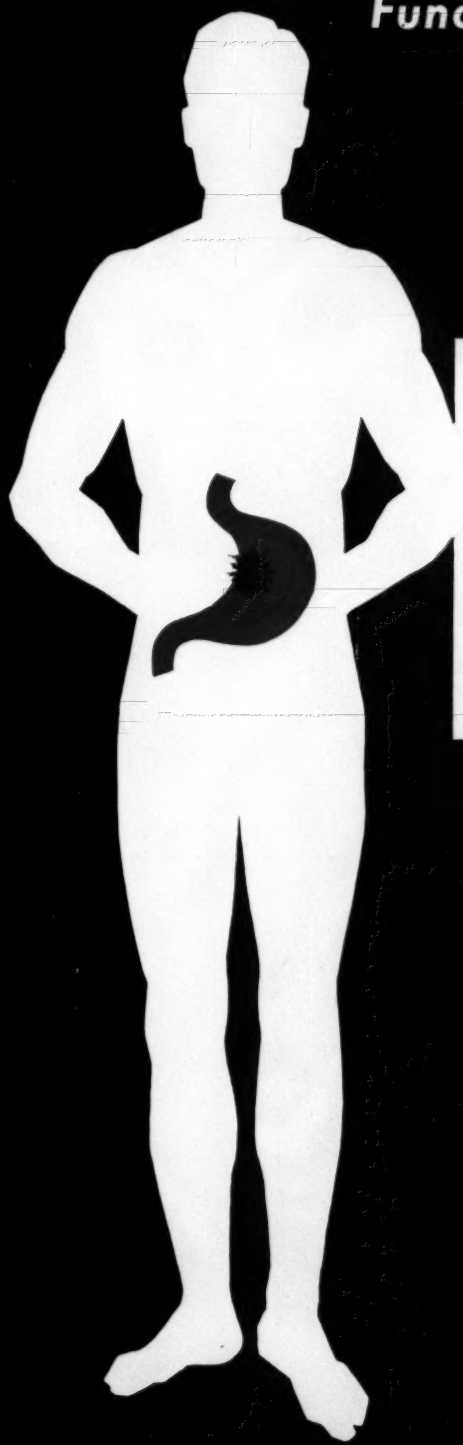
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Potent ANTISECRETORY • ANTICHOLINERGIC • SEDATIVE

Each tablet contains:

Monodral bromide	5 mg.
Mebaral	32 mg.

Dependable control of hyperacidity and hypermotility. Spasmolysis. Prompt and prolonged pain relief. Tranquillity without drowsiness.

Peptic ulcer, 1 or 2 tablets three or four times daily. Other gastro-intestinal disorders, 1 tablet three or four times daily.

Bottles of 100 tablets.

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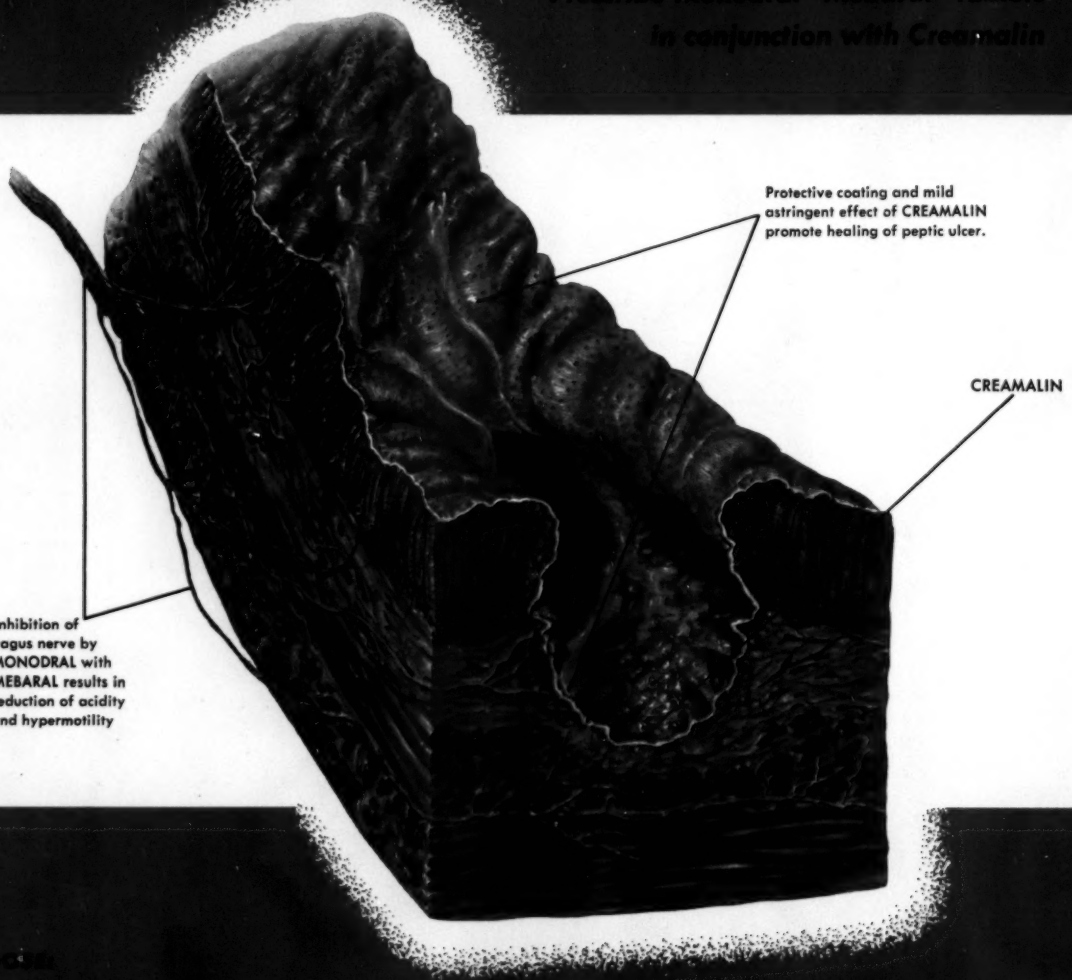
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PIONEER ALUMINUM HYDROXIDE GEL

FAST ACTING REACTIVE GEL

For best results in PEPTIC ULCER

Prescribe Monodral®-Mebaral® tablets in conjunction with Creamalin



DOSE

From 2 to 4 teaspoonfuls Creamalin liquid or from 2 to 4 Creamalin tablets (well chewed) every two to four hours, with a small amount of water or milk.

Creamalin liquid — 8 and 16 fl. oz.

Creamalin tablets — bottles of 50 and 200.

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Paris, too, knows and uses Pentothal...

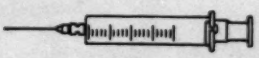


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Pentothal Sodium has been in constant use for 23 years. In that time more than 2500 reports have been published on Pentothal, covering nearly every type of surgical procedure—*making Pentothal unmistakably the world's most widely studied intravenous anesthetic.* Reflected in these years of use and volumes of reports is a record unsurpassed for safety, effectiveness and versatility of use in intravenous anesthesia. Do you have the literature? Abbott

PENTOTHAL® Sodium



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706139

NEW...



Each Multiple Compressed Tablet of MEPROLONE provides the inseparable antiarthritic, antirheumatic benefits of:

1. *Prednisolone buffered*—the newest and most potent of the "predni-steroids" for prompt relief of joint pain and arrest of the destructive inflammatory process.

2. *Meprobamate*—the newest and safest of the muscle-relaxant tranquilizers for profound relaxation of skeletal muscle in spasm.

Tolerance to this combination is good because there is little likelihood of sodium retention, potassium depletion or gastric distress with buffered prednisolone, and meprobamate rarely produces significant side effects in therapeutic dosage.

An additional important therapeutic benefit, often overlooked, stems from the tranquilizing action of meprobamate. This component of MEPROLONE relieves mental tension and anxiety so often manifest in arthritics, making them more amenable to other rehabilitation measures.

INDICATIONS: A wide variety of conditions, in which four symptoms predominate: *a*) inflammation *b*) muscle spasm *c*) anxiety and tension *d*) discomfort and disability; i.e., rheumatoid arthritis, rheumatoid spondylitis (Marie-Strümpell disease), Still's disease, psoriatic arthritis, osteoarthritis, bursitis, synovitis, tenosynovitis, myositis, fibrositis, fibromyositis, neuritis, acute and chronic low back pain, acute and chronic primary and secondary fibrositis and torticollis, intractable asthma, respiratory allergies, allergic and inflammatory eye and skin disorders (as maintenance therapy in disseminated lupus erythematosus, periarteritis nodosa, dermatomyositis and scleroderma).

Therapeutic benefits of MEPROLONE compared with traditional antiarthritics.

	relieves pain	suppresses inflammation	relaxes muscle	eases anxiety	imparts sense of well-being
Salicylates	✓	✓			
Muscle relaxants			✓[1]		
Tranquilizers				✓[1]	
Steroids	✓	✓			✓
MEPROLONE	✓	✓	✓	✓	✓

1. Meprobamate is the only tranquilizer with muscle-relaxant action.

SUPPLIED: Multiple Compressed Tablets in bottles of 100 in two formulas as follows: MEPROLONE-1—1.0 mg. of prednisolone, 200 mg. of meprobamate and 200 mg. of dried aluminum hydroxide gel. MEPROLONE-2—provides 2.0 mg. of prednisolone in the same formula.

NO OTHER

ANTIRHEUMATIC

PRODUCT

PROVIDES AS MANY

BENEFITS AS

MEPROLONE'

MEPRO PREDNISO | BAMATE LONE, *buffered*

THE ONLY

ANTIRHEUMATIC,

ANTIARTHRITIC

THAT SIMULTANEOUSLY

RELIEVES:

1. MUSCLE SPASM
2. JOINT INFLAMMATION
3. ANXIETY AND TENSION
4. DISCOMFORT AND DISABILITY

MSD

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DIVISION OF MERCK & CO., Inc. PHILADELPHIA 1, PA.

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WHY SENSITIZE

in topical and ophthalmic infections

USE 'POLYSPORIN'®

brand

POLYMYXIN B—BACITRACIN OINTMENT

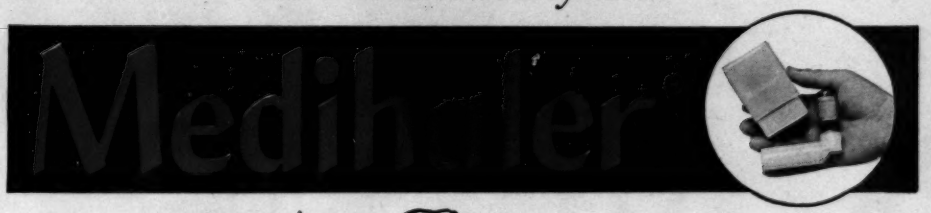
to insure broad-spectrum therapy with minimum allergenicity

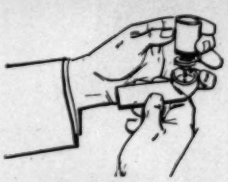
For topical use: in ½ oz. and 1 oz. tubes.
For ophthalmic use: in ⅛ oz. tubes.

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MILLIONS OF ASTHMATIC ATTACKS

have been aborted faster...more effectively... more economically with





SIMPLE TO USE



CONVENIENT



SUITABLE FOR CHILDREN, TOO



SLIPS INTO POCKET OR PURSE

Automatically measured dosage and true nebulization...nothing to pour or measure...One inhalation usually gives prompt relief of acute or recurring asthmatic attacks.

Medihaler-Epi replaces injected epinephrine in urticaria, edema of glottis, etc. due to acute food, drug or pollen reactions...Each 10 cc. bottle delivers 200 inhalations.

IN ASTHMA PRESCRIBE EITHER

Medihaler-EPI® Riker brand epinephrine U.S.P. 0.5% solution in inert, nontoxic aerosol vehicle. Each measured dose 0.12 mg. epinephrine. In 10 cc. bottle with measured-dose valve.

Medihaler-ISO® Riker brand isoproterenol HCl 0.25% solution in inert, nontoxic aerosol vehicle. Each measured dose 0.06 mg. isoproterenol. In 10 cc. bottle with measured-dose valve.

Note: First prescription for Medihaler medications should include the desired medication and Medihaler Oral Adapter (supplied with pocket-sized plastic carrying case for medication and Adapter).

The Medihaler Principle

is also available in Medihaler-Nitro™ (octyl nitrite) for the rapid relief of angina pectoris ...and Medihaler-Phen™ (phenylephrine-hydrocortisone-neomycin) for lasting, effective relief of nasal congestion.

Riker LOS ANGELES



She Helped a Burglar make his Getaway

Her home was in a part of the city where anything could happen



WAKING ONE MIDNIGHT, she surprised a burglar in her room. As he leapt for the window, she stopped him. "You'll be hurt. Go down by the stairs and let yourself out."

Calm, kind, and acutely intelligent, she had long ago learned to stay human in emergencies—by living where emergencies were routine, in the heart of one of Chicago's poorest immigrant neighborhoods.

Here she had settled down to her life work—helping people. No sociologist or social worker, she left it for others to make this a science. To her, it was an art. An art she practiced so beautifully that, eventually, while she was loved around Halsted Street, she was admired around the world.

When, in 1935, Jane Addams of Hull House died, her little grandniece, seeing hundreds of children among the mourners, asked, "Are we all Aunt Jane's children?"

In a sense, we all are. For the work Jane Addams did and the lessons she taught still help us all. And they prove magnificently the fact that America's greatest wealth lies in Americans.

It is the character and abilities of her people that make this country strong. And it is these selfsame people who make our nation's Savings Bonds one of the world's finest investments. For in U. S. Savings Bonds your principal is guaranteed safe to any amount—and your interest guaranteed sure—by the government that represents the united strength of 168 million Americans. So for your family's security, buy Savings Bonds. Buy them at your bank or through the Payroll Savings Plan at work. And hold on to them.

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— John Ruskin



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a new dosage form

Compazine★ *Ampuls*

for immediate control of nausea and vomiting when oral administration is not feasible

In 98% of cases treated with 'Compazine' Ampuls during clinical trials, a single intramuscular dose completely stopped nausea and vomiting or reduced its severity enough to permit tablet administration.

Dosage: An initial dose of 5 to 10 mg. (1 to 2 cc.) should be injected *deeply* into the upper outer quadrant of the buttock. This may be repeated if necessary at intervals of 3 to 4 hours.

For further information, see S.K.F. literature.

Available: 2 cc. (10 mg.) ampuls in boxes of 6 and 100. 5 mg. tablets in bottles of 50 and 500.

'Compazine'

the outstanding antiemetic with minimal side effects

Smith, Kline & French Laboratories, Philadelphia

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